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Editorial

ON STUDYING AROUND EXAM TIME

It has always been a great wonder to see students dropping out of their classes and staying away from clinics, starting a month before, and continuing right up until examinations. When you ask them "Why" you get the reply: "I can't go to school, I'm too busy studying!" And this just doesn't apply to exam time, it applies to many tests throughout the year: we stay away from Physiology lab. to study Anatomy, and stay away from Anatomy lab. to study Physiology; from Medicine and Surgery clinics to study Pathology, and from Pathology lab. to study Medicine and Surgery. This is indeed a vicious circle.

Of course it is very commendable to be so interested in studying. Let us, however, analyze this bookishness; what is the prime mover that makes us such book lovers. We all know the answer: it is not that we are interested in real learning, but that we desperately desire to accumulate a lot of half truths, sufficient to pass that great evil of a student's life—the examination.

There is a certain type of student that thinks in terms of examinations all the time, but most of us begin to think of them only in that last bad month, when we are willing to sacrifice our sleep, our good clinics and labs, even our food and girl friend to hurdle the inevitable. This is mass hysteria, nothing less. For the very borderline student who sees a thousand dollars go "down the drain" if he fails, it is perhaps commendable for him thus to try to "fool" the examiner; but

for the better than average student, it is nothing less than criminal to let the bogey of examinations keep him from leading his normal life, and from attending the utmost number of clinics and labs.

Examinations certainly defeat any purpose they have if they make such cravens of us, make us forget all about Medicine in a great struggle for marks. If the final examinations are of this great importance in the eyes of the examiner, something should be done to make them less so.

Why can't we continue our normal lives up until a week before the exams, and then "shoot the bolt" in that week, which is generally set aside for studying anyway. And as for tests throughout the year, we shouldn't stay away from classes a week before taking an Anatomy exam. The Professor himself would be the first to "cry halt" to such a procedure.

So let us gird up our loins, let us be more interested in man, than in one of his diabolical machinations—the exam!

THE TREATMENT OF MUSCLE PARALYSIS IN POLIOMYELITIS

By A. B. LeMESURIER, M.B.

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Acute anterior Poliomyelitis is a virus disease, in which, after the acute stage, practically the only feature is a varying extent of muscle paralysis. Any damage, at all lasting, done by the virus is limited almost entirely to the anterior horn cells of the spinal cord. There is nothing really wrong with the paralyzed muscle fibres themselves. They are paralyzed because the motor nerve supply to them is cut off at the lesion in the spinal cord. Their recovery depends on the recovery of the nerve cells, and the paralyzed muscles can regain power only when the damaged nerve cells have recovered sufficiently to bring motor stimuli to them. Some of these damaged nerve cells have been only slightly damaged and recover very rapidly, some of them take a long time to recover, and some of them are so badly damaged that they do not recover at all. Theoretically, the treatment should be directed chiefly towards the point of damage, that is, towards the spinal cord, but unfortunately there is no known method of treatment that will in any way hasten or improve the recovery of these damaged nerve cells. The ones that are temporarily damaged will recover in their own time, and the ones that are badly damaged cannot be made to recover by anything that we can do to them. The only thing that we can do in the way of treatment has therefore to be directed towards the paralyzed muscles, and is designed to keep them in such a condition that they will be able to resume their function as soon as the nerve supply to them is restored.

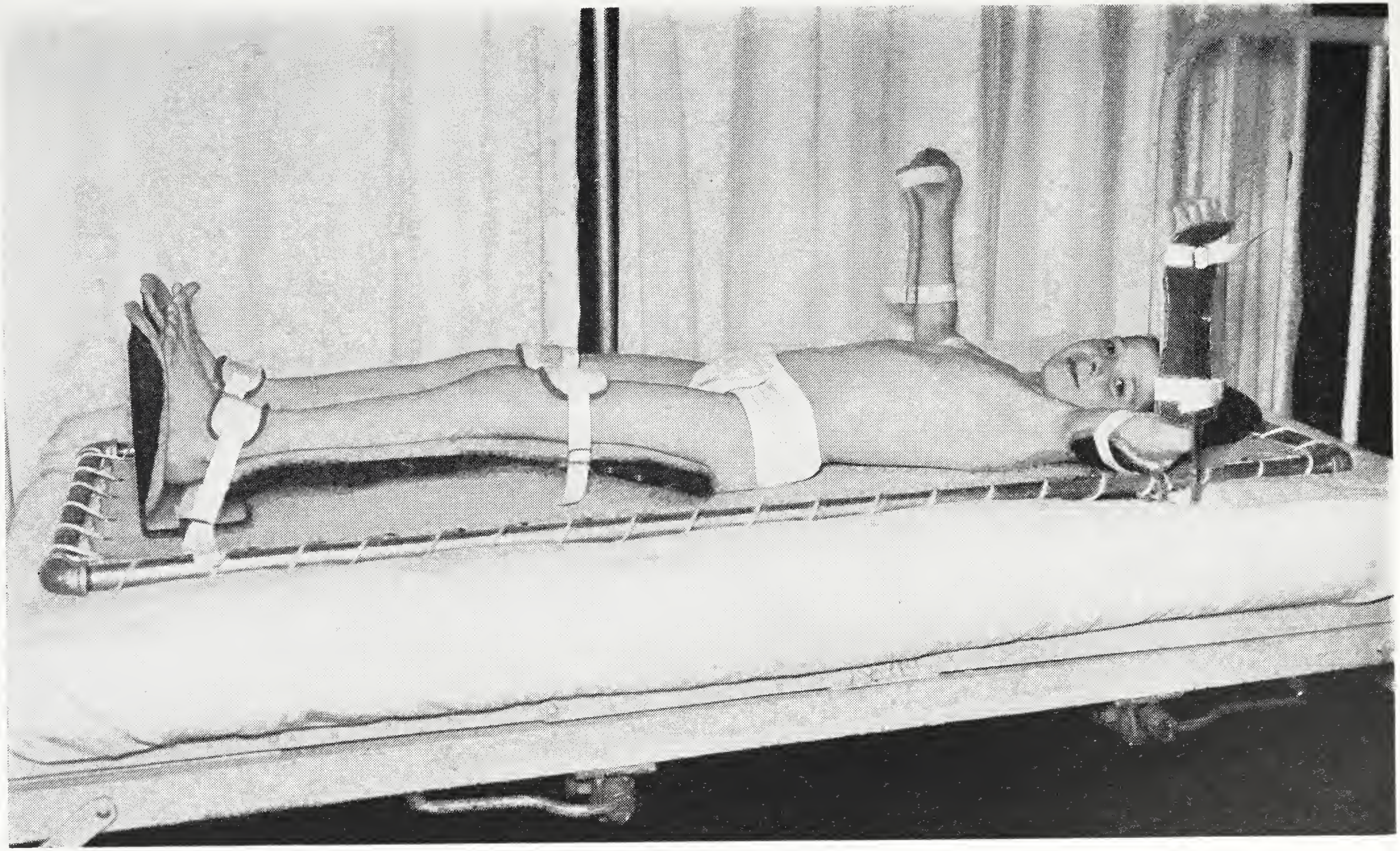
The way to keep these paralyzed muscles in condition to resume their function is to keep up their nutrition as well as possible, and to avoid anything that does them harm. There are two main things that will prevent a muscle from regaining function when the nerve cell has recovered, and these are over-stretching and over-fatigue. A muscle that has been held for a long time in the over-stretched position will not regain power when the nerve supply to it is restored, and even frequent over-stretching will greatly delay the return of power. To avoid this over-stretching, the paralyzed muscle should be held constantly in the relaxed position. Over-fatigue of a muscle that is regaining power will greatly delay its improvement, and, if the over-fatigue is repeated several times, the power may disappear, and, if it returns at all, will return slowly. If a muscle is just beginning to regain power it is very easily over-fatigued, and it is a mistake to encourage the patient to perform movements that are just beginning to appear. If a limb is allowed to be used, the patient will use, in that limb, any muscle power that is present, and, if a limb is allowed to be used freely, any muscle that is recovering will soon become over-fatigued and its recovery greatly delayed. To avoid this easily produced over-fatigue, the activity of a limb should be greatly restricted, particularly during the early stage of recovery and, in the lower limbs, any walking, even with a supporting brace, should be delayed until the muscles have regained almost as much power as they are going to regain.

These two harmful factors—over-stretching and over-fatigue—can best be avoided by the use of splints. To prevent the over-use of a limb, it matters very little in what position it is splinted as long as it is kept reasonably quiet, but in order to prevent over-stretching of the muscles, the position in which the limb is splinted is important. It is impossible to keep all the muscles relaxed in the one position, because, if one muscle is held relaxed, its opposing muscle is over-stretched. However, there are certain muscles that, if paralyzed, are almost certain to be

held constantly in the over-stretched position unless the limbs are splinted. This applies particularly to the deltoid, the dorsiflexors of the foot and the extensors of the knee and hip. These muscles are the ones most frequently paralyzed, and their recovery is of the greatest importance to the patient. The wisest procedure, therefore, is, when a part is affected, to apply as soon as possible splints that hold these particular muscles in the relaxed position. If the muscles of the arm are affected, the shoulder should be kept in abduction. If the muscles controlling the trunk or abdomen are affected, the patient should be kept continually flat on his back and not allowed to sit up. If the muscles of the hip are affected, he should be kept flat on his back with the hip extended and the whole limb prevented from falling into internal or external rotation. If the muscles controlling the knee are affected, the joint should be kept in almost complete extension, and if the muscles controlling the foot are involved, the foot should be held at a right angle to the leg. In some cases it may become evident, at a later date, that these positions are causing over-stretching of muscles that are badly paralyzed and whose recovery is important, and the position of the splint should therefore be changed accordingly. However in almost every case the positions described above are satisfactory in the early stages of the paralysis, and in very few cases is it necessary to bring about any change.

The actual splinting requires a good deal of care. It is almost impossible, by means of improvised apparatus made by pillows or sand bags, to keep the shoulder constantly in abduction or the foot at a right angle to the leg. Plaste splints can be made, or a splint-maker can make a special splint for each patient, but both these methods are either difficult or slow. During the severe epidemic in Ontario in 1937 a method was worked out to overcome this difficulty of getting splints that would fit each patient. The positions, described above, to hold the important muscles relaxed, were adopted, and splints were made that would maintain these positions, and were made in enough different sizes to fit any patient. The right size of splint can be obtained from measurements simple enough to be taken by anyone. Only three appliances are necessary—the arm splint, the leg splint and the Bradford frame. The arm splint holds the shoulder abducted, the elbow at a right angle, the fore-arm supinated, and the wrist extended. It is fastened to the Bradford frame and cannot be used without the patient lying on his back on this frame. To obtain the right size of arm splint, only one measurement is required—the length of the fore-arm as measured from the tip of the elbow to the web of the thumb. The leg splint holds the knee in almost complete extension and the foot at a right angle to the leg. With the patient lying on his back on the Bradford frame, the hip is held extended and the limb is prevented from rotating in either direction. To obtain the right size of leg splint only one measurement is necessary—the distance between the centre of the patella and the sole of the foot. The use of the Bradford frame is necessary when either limb is splinted, and its chief object is to keep the patient flat on his back, with his hips extended. Only one measurement is necessary to obtain the proper size of frame, that is the height of the patient.

These standard appliances have been very satisfactory, and their use provides the simplest way of maintaining the required position. Besides preventing the over-stretching of the paralyzed muscles and the over-use of the recovering muscles, the use of splints also prevents deformities. In Poliomyelitis, deformities are common, chiefly as the result of the uneven distribution of the paralysis. It often happens that the muscles that move a joint in one direction are completely paralyzed whereas the muscles that move the same joint in the other direction are practically normal. If nothing is done to prevent it, the lesser paralyzed of the two opposing muscles will always be held in the contracted position and in time will become structurally shortened, with the formation of a deformity which cannot be corrected passively. The



PATIENT ON BRADFORD FRAME WITH ALL FOUR LIMBS SPLINTED IN STANDARD POSITIONS

occurrence of these deformities is prevented if the paralyzed muscle is held relaxed in a splint.

Splinting of the paralyzed limbs prevents over-stretching of the paralyzed muscles, over-use of the recovering muscle and the appearance of deformity. However there are other things necessary to keep the muscle in proper condition. The paralyzed muscles become in time very much atrophied, the circulation of the whole limb becomes sluggish, and the joints may become stiff, and all these things can be lessened by physio-therapy. In the early stages, at any rate, physio-therapy should be limited to massage and passive movement. These can of course be best done by a trained physio-therapist, but this is not always possible. The patient's parents are apt either to neglect or over-do the treatment, but if they are first shown how to carry it out properly, and are supervised at intervals, they can do it, on the average, fairly well.

Massage of course does not restore power to a muscle until the nerve supply is restored. Its main object is to stir up the sluggish circulation, and if it is done properly it will considerably improve the circulation and general condition of the whole limb, and will lessen considerably the atrophy of the muscles. If it is done too heavily, or continued too long, it produces much the same signs as fatigue, and should therefore be done lightly, and ten minutes a day should be long enough for each limb. Passive movement is intended to keep the joints from becoming stiff, but there is no point in overdoing it. It is a mistake ever to force a joint like the knee, which is the most likely joint to become stiff, into extreme flexion, because this can only be done by over-stretching the quadriceps muscle, which is frequently paralyzed, and is a muscle whose recovery is important. As a general rule, moving all the joints of an affected limb through about half their normal range once or twice a day is enough in the way of passive movement.

The usual history of a case of Poliomyelitis is that in August, September or October the child becomes sick, with some elevation of temperature and symptoms that vary very much in character and severity. About half these cases recover without any muscle paralysis, and in these it is difficult to make a definite diagnosis except by an increase in the cell count of the cerebro-spinal fluid. In the cases in which paralysis develops, the muscle weakness is first noticed about the second or third day, and increases for the next two or three days and then stops increasing. The affected muscles are sore and pressure over the muscle bellies, or stretching them by moving the joints, causes pain. The paralysis is of the lower motor neurone type, flaccid in character, with absent or diminished reflexes, with slowly appearing atrophy and without any sensory change. The extent of the paralysis varies from slight weakness of a single muscle to a complete paralysis of all the muscles of all four limbs and of some of the muscles of the trunk. There is nothing symmetrical about the distribution of the paralysis, and almost any striated muscle may be affected, although some muscles are more commonly affected than others.

As soon as the paralysis is detected the affected limb should be kept quiet, and the sooner it is splinted the better. The patient has much less discomfort when the limb is splinted in the proper position within the first week than if it is done at a later date. Massage and passive movement cannot be commenced until the muscle soreness has almost disappeared, which is usually not until three weeks from the onset. The treatment should then be carried out more or less as a routine. The affected limbs should be kept continually in the splints, and in most cases the patient should be kept flat on his back. The limbs may be taken out of the splints twice a day, once for the necessary washing and care of the skin, and once for massage and passive movement, but on each occasion the limbs should not be used too much and no muscle should be over-stretched. For the rest of the time the patient should be reasonably quiet and should be kept occupied and amused, which in small children is difficult.

In most cases some return of muscle power takes place, starting about a

month after the onset. The amount of recovery varies from only a slight improvement to an almost complete recovery of all the affected muscles. The same course of treatment—relaxation of the muscles by splints and the use of physio-therapy—should be continued until one of two things happens, either until enough recovery has taken place in all the muscles to warrant increased activity, or until enough time has elapsed that there is no hope of further recovery.

If the paralyzed muscles are recovering, active movement is started very slowly. Exercise is beneficial to a muscle only when it stops short of producing fatigue, and these recovering muscles are very easily over-fatigued. Over-fatigue will stop their recovery, whereas improvement will continue if they are kept at rest in the relaxed position by a splint. The advantage of exercise can be obtained later when the recovery is well advanced, and exercise can be used then with less chance of doing harm. For instance if the muscles of a lower limb show signs of recovering, the patient is not immediately allowed to walk. The constant fixation of the limb in a splint is continued until occasional tests show that power in the muscles is improving and recovery is fairly well advanced. The splint is then removed for a short time, at first for about fifteen minutes each day. and the child encouraged to kick the whole leg about rather than to use the particular muscles that are recovering. If there is no decrease in the power, the time and amount of active movement are gradually increased. When he first gets up it should only be for a short time each day, slowly increasing if there are no signs of fatigue. When a patient is up and walking, fatigue can be detected by an increasing limp. The recovering muscles should be protected against over-stretching and over-fatigue for a long time, and these patients should wear their splints at night and lead a quiet life until their recovery is far advanced.

In the cases that are not going to recover completely it is often difficult to tell how long it is worth while to continue treatment by splints. In the early stages there is no way of predicting how much recovery there will be, or how rapid it will be. Most of the recovery that is going to take place takes place during the first six months, but, while improvement after that becomes slower, it may continue for two years or even longer, if over-stretching and over-fatigue are prevented. Time means little to these children but even a slight improvement in muscle power means a good deal and, if there is any chance of further improvement, it is well worth while continuing the treatment. If, therefore, at the end of a year from the onset of the paralysis, there has been absolutely no improvement during the previous three months, and if treatment has been carried out efficiently. it is perhaps time to abandon the splints, but if there has been the slightest increase in power, or if the treatment can be improved in any way, it is worth while continuing it until two years from the onset or, in some cases, longer.

When the splint treatment is finally given up, it does not mean that the child is going to be incapacitated for the rest of his life. It means that no further recovery can be expected in the paralyzed muscles, and the amount of disability depends on the extent of the permanent muscle paralysis. It is surprising how well some of these patients get about with fairly extensive paralysis, and, at this stage, the treatment should be directed towards making the most of what muscle power remains. Many of the patients need no further treatment beyond exercise to improve the muscles that were never paralyzed or have recovered, and the massage should be continued until the affected limbs are being used a reasonable amount each day.

In the more severe cases some further treatment may be necessary. The usefulness of a limb can often be increased considerably by the wearing of a brace designed to hold a joint, which has lost its muscle control, in the most useful position. The two most useful braces are, the long leg brace which holds a flail knee extended and stops the patient

from falling when walking, and the stop-joint ankle brace which holds a drop-foot up at a right angle. These are worn fixed to the shoe and strapped to the limb and, beyond the inconvenience of their weight, are often a great help. In many of the severe cases operations are required. These are done for two purposes, to correct a deformity or to stabilise a joint in the most useful position. A deformity can usually be corrected by lengthening the tendon of the shortened muscle, such as the lengthening of the tendo Achilles in an equinus deformity. However, lengthening the tendo Achilles will not hold the foot up at a right angle if the dorsiflexor muscles are paralyzed, and, in addition to the lengthening of the tendon, a brace is worn or some stabilising operation is done. The usual stabilising operation in the lower extremity is an arthrodesis, an operation intended to ankylose the joint, and done by removing the articular cartilage and holding the raw surfaces of the two bones together in the desired position until they unite, as a fracture does. These arthrodesis operations are very satisfactory in the foot, where they are used to hold a varus or valgus deformity corrected. They are being done more and more frequently in the ankle joint to hold a drop-foot up, and are occasionally done in the knee and hip. Their main advantage is that they allow braces to be dispensed with. They leave the joint permanently stiff and of course should not be done until all hope of muscle recovery is past. Other operations such as muscle transplantations or tendon fixations are sometimes done with advantage, but in the lower extremity they seldom are of much help. Many of the patients with extensive paralysis require several operations, but there are extremely few that cannot be made to get about fairly well by themselves.

"Domestic Electropathy"

"Let us illustrate how this invaluable and mighty agent works in some of its applications, restoring dead or nearly dead parts of the body to life and health, as in the case of the torpid or inactive liver, stomach, bowels, etc. For example:— Hit your finger instead of the nail you intended to drive, and it crushes the finger and nail into a discoloured mass; the nail turns black and half of it becomes as if it were dead, and shows that it is about to separate from the finger. You expect nothing less than to lose the nail. The pain in this case is intense until the nail is actually loose and begins to come off and a new one forms. But while the nail is yet but half alive & half dead, apply electricity with one of our Improved Batteries and you will quickly remove the pain and reduce the inflammation. You will hardly believe that the black dead part can be restored. Yet so it is; it has been done and may always be effected, provided suppuration has not taken place. So surely as there is some life left in the part of the nail we can with our system of electropathy, gradually, so act upon the living tissues as to bring back the dead part to life again and restore the whole nail and finger. In the same way we can act upon the liver, which may be half-dead; the larger portion of it may be as torpid and inactive as a piece of sole-leather, and no drug treatment capable of restoring it, yet with the proper use of electricity we can bring the organ back, like the finger, to an active and healthy state."

A Handbook and Guide to Domestic Electropathy
By Professor Vernoy,
Electro-TheraTherapeutic Institute,
Jarvis St., Toronto, 1884.

NOTES ON THE IMMUNOCHEMISTRY OF SERUM

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Serum contains a complex of proteins. Generally these are considered to be serum albumin and serum globulin, or more correctly serum albumins and serum globulins, the latter consisting partly of true globulin (euglobulin), soluble in neutral salt solutions but insoluble in water, and partly of material soluble in water and hence called pseudoglobulin. But it has been evident for a long time that this simple classification requires modification and expansion. Instead of 3 protein fractions in normal serum one can now easily extend the list to 13, and this figure still would not include enzymes and hormones, known to be present in serum, though in relatively small amounts. Any immunochemical consideration of serum should therefore be based on some of these newer successful fractionations of the protein constituents of both normal and immune serum.

Various chemical procedures have been long in use for the fractionation of protein mixtures, and are still used today with increasing success. Of these may be mentioned the "salting out" processes; the use at various pH values of different concentrations of ammonium sulphate, sodium sulphate or potassium phosphate, and also the "salting in" processes, wherein protein precipitates are fractionally extracted with dilute buffer solutions of the required ionic strength and hydrogen ion concentration to dissolve one portion and leave the remainder undissolved. Recently fractionation of serum proteins by means of various concentrations of alcohol at low temperatures has been practised with considerable success. Dialysis and electrodialysis are methods of especial value in the separation of albumins and pseudoglobulins from the euglobulins. During the last few years considerable advance has been made in protein fractionation by means of electrophoresis and by very high speed centrifugation. If an electric current be passed through a solution containing electrically charged particles, such particles will migrate to one or other of the electrodes at a characteristic rate. Of late years this behaviour of proteins has been extensively studied in the Tiselius electrophoresis apparatus. The rate at which any particular protein migrates in an electric field has been found to depend not only upon the ionic strength, temperature and hydrogen ion concentration of the buffer solution in which it is dissolved but on its own size and electric charge. When therefore normal serum, mixed with a suitable buffer, is examined in the apparatus it is found that the globulin proteins move with at least 3 different velocities and these fractions are now called, as by Tiselius, α, β and γ globulins, in order of their relative mobilities. Each of these fractions can be separated, by dialysis, into pseudoglobulin and euglobulin fractions. All these fractions have been obtained also by the chemical methods mentioned above. By these methods the fractionation has been further extended; for example, the γ euglobulin fraction of horse serum has been sorted out into four different components. None of the serum globulins has been crystallized. The albumins are readily distinguished from the globulins in the Tiselius apparatus because of their greater mobility. It might be mentioned here that two crystalline albumins of horse serum have been isolated by fractional precipitation with ammonium sulphate; one is free of carbohydrate, the other contains a polysaccharide as part of the albumin molecule. These two albumins have different electrophoretic mobilities. It should be appreciated that the electrophoresis apparatus is used on occasion to effect a separation of the different fractions of serum proteins (or, of course, other protein mixtures) and at other times to test the homogeneity of fractions obtained by chemical precipitation methods. Under suitable conditions a contaminating protein present to the extent of only 5 per cent can often

be detected. The Svedberg ultracentrifuge also has been used for the above purposes of fractionation and testing of homogeneity of serum proteins. Different proteins sediment at different speeds depending upon their size and shape. It has been calculated from the sedimentation constants (obtained by the ultracentrifuge) in conjunction with diffusion constants (which also depend upon size and shape) that serum contains proteins having molecular weights of 70,000, 150,000 and 900,000. Those of lower molecular weight are the albumins; the bulk of the globulin fractions, whether pseudo- or euglobulins, have a molecular weight of 150,000; the high molecular weight fraction is formed as part of the γ euglobulin fraction.

These physical and chemical methods have been used in analyses of normal sera from different species and of immune sera (see R. Hare, This Journal, 1938). Thus sera from man, horse, cow and rabbit have been examined. The accompanying table, taken from a recent publication of E. J. Cohn and his coworkers shows the distribution of the different protein constituents of horse serum. Essentially the same type of general pattern is obtained with sera of other species, but there are differences in the amounts of the various fractions and in the relative proportions of pseudo- to euglobulin. Here it may be mentioned that the Tiselius apparatus has been used in investigating changes in the serum proteins under various pathological conditions. Thus in a number of febrile conditions (e.g., pneumonia) an increase in the amount of the α globulin fraction has been noted and in some other conditions (e.g., nephrosis, obstructive jaundice) increases in the β globulin fraction have been observed. Such determinations may become the routine to supplant or supplement the customary "albumin/globulin ratios."

We are now at the stage of scientific endeavour when the different immunological constituents of serum may be investigated as discrete substances. Thus there has been accomplished the successful isolation from serum in a high degree of purity of antibodies (to mammalian and bacterial proteins) including antitoxins, and also the isolation of components of complement.

Antibodies are proteins synthesized by cells (what cells are concerned is still a matter of dispute) under the stimulus of a foreign protein, called the antigen. For obvious technical reasons the antibodies that have been studied to date are those found in the serum. It seems legitimate to regard antibodies as globulin proteins modified at the time of their synthesis by the influence of the foreign antigenic protein, rather than as direct modifications of this antigenic protein itself. It would appear that these modified globulins (antibodies) differ from the normal serum globulin for the most part in a new spatial arrangement of amino acids within the

Serum Proteins of the Horse

Concentration of ammonium sulfate % of satn.	Type of protein precipitated	% of total protein pptd.	% of protein pptd. Water soluble	Water insoluble
0.34	Largely γ-globulin	20	71	29
0.40	γ-, β-, α-globulin	15	67	33
0.50	β-, α-globulin, muco-globulin	14	94	6
0.62	Largely crystalline albumins	32	98	2
0.68	Crystalline albumins, haemocuprein, choline esterase, glycoprotein, phosphatase	14	99	1

protein molecule, rather than by any difference in the type of amino acids or of the number of the free acid or basic groupings in the molecule. Serological specificity appears to depend then very largely on the particular arrangement of the amino acids in the molecular framework. This applies to antigens and to antibodies.

Bearing in mind the multiplicity of possible antigens in serum (i.e., if the serum of one animal be injected into an animal of another species), one can appreciate the possible complexity of the resulting antibody response. In an effort to make the analysis of the responses simpler, a number of immunologists have for years urged the use of what they called pure proteins in the place of such complexities as in serum. But it is increasingly difficult to speak of a pure protein. Crystalline egg albumin of the hen, which some have advocated because it appears to be homogeneous, has recently been shown to consist of at least 3 components, as judged by its behaviour in the Tiselius electrophoresis apparatus. But it still remains to be shown whether these different egg albumin components behave as distinct antigens, for it is possible that the spatial distribution of amino acids determining serological specificity may be identical in all. The same type of consideration applies to the newer fractions of serum protein. Older investigators, who were able to effect a sufficiently good separation of the 3 main fractions, albumin, pseudoglobulin and euglobulin, were able to demonstrate that all three fractions, when employed to sensitize guinea pigs, possessed different antigenic properties. The antigenic properties of the components of these main fractions have yet to be determined.

The 3 successive serum rashes which occur sometimes in persons a few days after receiving an injection of immune horse serum have been attributed to these same protein fractions. In the development of sensitization (see H. H. Varty, This Journal, 1939) it appears that different animal sera vary greatly in their capacity as antigens in the tissues of different species, including man. It is generally agreed that the antigenicity of the globulin protein is greater than that of the albumins; the degree of sensitization is greater and the time necessary for the acquirement of sensitization shorter. Some workers consider the pseudoglobulin to be more strongly antigenic than the euglobulin fraction. This is the rationale of the procedure in which the albumin fraction of beef serum (readily obtained in large quantities) has been prepared and used clinically, on a small scale, as a human blood substitute in the treatment of shock. In spite of some evidence that beef serum proteins are of relatively low antigenicity towards man, especially the albumin fraction, there is still the danger of the development of a sensitization and steps are being taken to modify, chemically, the beef albumin molecule in an effort to change what antigenic specificity it possesses, and so prevent any untoward reaction when next the patient asks for and obtains his beefsteak rare!

Examined in the electrophoresis apparatus the normal serum of an animal shows no essential differences from serum, of the same species, but containing antibodies. There is a striking exception to this rule; horse serum containing antipneumococcus antibody shows a new serum component with a mobility between that of the β and γ globulins. Rabbit antipneumococcus serum possesses no such new component, for the antibody has been shown to migrate with the γ globulin fraction, which may be much larger than usual. It has been shown that whereas the antipneumococcus antibody isolated from immune horse serum has a molecular weight of 900,000, the corresponding antibody of the rabbit serum has a molecular weight of 150,000. In this fact may lie the reason for the greater efficiency of rabbit antibody than horse antibody in the treatment of patients with pneumonia. The pig and cow also elaborate an antipneumococcus antibody of 900,000 molecular weight. Undoubtedly the γ globulin component is the fraction of serum which contains most antibodies. Thus if a rabbit is injected repeatedly with a solution of

egg albumin, antibodies are formed in the animal and their presence in blood can be demonstrated because they are precipitated from a sample of serum of the animal when mixed under proper conditions with some of the antigen (egg albumin). In one animal the γ globulin component of serum was isolated and it was found that 80% of it was specifically precipitable, showing that the bulk of this component of serum was composed of the antibody. Heidelberger has shown that this reaction between antigen and antibody is not one of adsorption, as formerly believed, but is a strictly chemical reaction in which the reactants participate in definite proportions.

The various bacterial antitoxins present in immune serum have been shown, similarly, to be components of the γ globulin fraction, and to be pseudo-globulins.

Circulating antibodies are also found in certain allergic conditions. When sera from persons allergic to ragweed pollen were examined in the Tiselius apparatus, they presented no differences from the normal. These sera when injected into the skin of a normal man passively sensitized the area around the injection since a later injection of ragweed pollen extract into this skin produced a wheal as when ragweed pollen extract is injected into the skin of the donor of the serum. The serum thus contained skin sensitizing antibodies and these were shown to be present solely in its γ globulin component. When a rabbit was sensitized to ragweed pollen extract, again the precipitating antibody was found in the γ component of its serum.

A definite link between the condition of allergy, as represented by a patient naturally sensitized to ragweed pollen, and the condition of anaphylaxis as represented by a guinea pig artificially sensitized to ragweed pollen by parenteral injection, was established by experiments conducted in this University by the late Dr. Caulfeild and his co-workers. The serum of the patient and of the guinea pig passively sensitized local areas of skin of a normal person, as shown by local reactions on a subsequent injection of the antigen into these areas. In the allergic man and in the sensitized guinea pig there were circulating antibodies which reacted in a similar way with the common antigens.

With this increasing control over the chemical investigations of antigens and antibodies the science of immunology, with its important direct applications to human welfare, will provide the student of medicine with a new host of details to tax his memory, but also a more substantial basis for general principles, correlating cause and effect, than is now possible.

"On Writing a Thesis"

"I intend no breach of courtesy, but this is a proper place to warn the student against skimming the prefaces and introductions of works for mottoes and embellishments to his thesis. He cannot learn anatomy by thrusting an exploring needle into the body. He will be very liable to misquote his author's meaning while he is picking off his outside sentences. He may make as great a blunder as that simple prince who praised the conductor of his orchestra for the piece just before the overture; the musican was too good a courtier to tell him that it was only the tuning of the instruments."

"The Contagiousness of Puerperal Fever"
By Oliver Wendell Holmes,
Medical Essays 1842–1882
The Riverside Press, Cambridge.

A PROBLEM IN DIAGNOSIS

By C. C. GRAY, M.D.

From the Medical Service, Toronto General Hospital

[Editor's Note: The Meds-at-Home is over, and our funds are getting low, so it is impossible to give away any more prizes. However, if anyone is still interested, send in your answers to the "Problem in Diagnosis Committee", not later than Feb. 25th, and the winning student's answer will appear in the following issue.]

A twenty-four year old woman, Miss R. R., entered the hospital complaining of loss of power in legs and arms and difficulty in swallowing.

This patient was in her usual good health until three weeks before admission when she complained of lower right abdominal pain. Her physician made the diagnosis of acute appendicitis and under spinal anaesthesia the appendix and a small right ovarian cyst were removed. (Pathology not known as the operation was performed elsewhere.)

Five days post-operatively she became irrational. She talked about people fighting in the hall, attempted to get out of bed, was quite confused and disorientated for about eighteen hours. At this time she first complained of weakness of the muscles of both shoulder girdles. This progressed so that she could not move her hands. Within forty-eight hours she was unable to sit up and her legs were definitely weak. The loss of power in her lower extremities progressed so that she was only just able to move her toes at the time of her admission.

She complained of pain behind both knees for two days before admission, but no paraesthesia.

For twenty-four hours prior to admission she was having some difficulty swallowing solid foods, but there was no regurgitation of fluids through the nose. Also, during this time her breathing became rapid and shallow.

The patient had had the usual childhood diseases, but the family and personal histories were irrelevant.

Physical Examination

This revealed a poorly nourished pale girl whose respirations were 44 per minute and temperature was 101 degrees F. The lips were dry and cracked, the tongue coated with a brownish colour. Examination of the chest revealed breathing to be almost wholly thoracic in type with little or no movement of the diaphragm on either side. There was a patch of blowing breath sounds with a few crackling rales at the lower right base. The abdomen showed a well healed recent midline scar.

She was a bright cooperative girl, but a little apprehensive. There was no evidence of delusion, hallucination or aphasia. There was bilateral facial weakness and both trapezii and sternomastoids showed definite weakness. There was no paresis of the palate.

The upper extremities showed a complete flaccid paralysis and in the lower extremities she was just able to flex her knees and to move her toes. The only sensory disturbance was marked muscle tenderness.

Tendon reflexes were absent throughout and plantar response was flexor in type.

Laboratory Investigation

Urinalysis—negative.

Blood—Hb. 81%. R.B.C.—4,600,000. W.B.C.—18,400 with 92% polymorphonuclear cells.

Lumbar Puncture—revealed clear fluid under normal pressure with cell count of 1 lymphocyte per cu. mm.

Pandy—faintly positive.

Wassermann in C.S.F. was negative.

Total protein—25.5 mgm. %.

Vital capacity was 500 c.c. (the best of 10 attempts).

Treatment

1. Bed in respirator.
2. Isolation precautions.
3. To be fed through a duodenal tube with sips of water only by mouth.

4. Dagenan to be given through tube in pneumonia dosage.
5. Special care to mouth.
6. Mist. Brom. and chloral, drams ii p.r.n.
7. Ophthalmic ointment, mercury bichloride 1/3000 to eyes.
8. Permanent catheter—irrigated with boracic acid solution q. 8 h.

Course

Her temperature fell to 99 degrees F. (mouth) in thirty-six hours and remained at that level for only twenty-four hours when it began to rise again to 100 degrees on the third day and to 101 degrees on the fourth day. The pulse rate remained elevated at 130 beats per minute, respiratory rate continued between 30 and 40 (when out of Respirator).

Vital capacity was increased to 750 c.c on her second day in the hospital.

During her last twenty-four hours muco-purulent material collected in the trachea and this was partially aspirated by means of a tracheal catheter attached to a suction pump. Eventually this fluid became too copious and death occurred as a result of tracheal obstruction.

No change in neurological findings were elicited during her days in the hospital.

WINNING STUDENT'S ANSWER TO PROBLEM IN DIAGNOSIS

By H. H. MacKINNON, '41

On the previous admission, this patient suffered pneumonia and empyema. From the absence of fever and other signs of inflammation, we can accept that the terminal illness was no inflammatory sequela to the previous disease. Examination in the O.P.D. on Oct. 21st revealed no sign of disease; beyond the fast pulse and a moderate increase in blood pressure, nothing abnormal was found. The diastolic blood pressure was raised relatively more than the systolic. She complained of belching and lower thoracic back pain. In the convalescent home the belching almost disappeared and the pain eased.

One month following this, she was admitted with marked hypertension and signs of increased intracranial pressure (congestion of the retina, raised C.S.F. pressure; headache and vomiting). The question may be asked which came first? From the former heightened diastolic pressure, and the moderately enlarged heart, we may conclude that the hypertension was essential in nature. It does not, of course, follow that the raised intracranial pressure was due the hypertension.

The raised intracranial pressure was not due to uremia, as the blood N.P.N. was only raised terminally; and the urine was more concentrated than is found in chronic nephritis. The C.S.F. findings were not indicative of meningitis. There are no localizing signs of primary brain or spinal cord tumor. Multiple secondary tumors in the brain might raise the pressure but would not cause the urinary findings. It is most probable that the rise in intracranial pressure was due to the hypertension. The hypertension also caused rupture of a cerebral vessel with subarachnoid or intraventricular hemorrhage, which prior to the puncture was not massive, as there were no reported signs of meningeal irritation.

Fishberg states that "in some cases of the malignant phase of essential hypertension, these consequences of increased intracranial pressure due to edema of the brain completely dominate the clinical picture, which simulates closely brain tumor".

The blood pressure as recorded was not remarkably high, but hypertensive patients are subject to fluctuations in pressure; the pressure also varies in different parts at one time.

The staring eyes have been noted in edema of the brain, chronic nephritis, and malignant hypertension.

The X-ray appearance of the spine was due to a combination of the scoliosis and the aortic pressure. The pain noted was due to increased stretching of an arteriosclerotic aorta with the rise in blood pressure.

The terminal pain in the right shoulder tip was due to diaphragmatic irritation, due to increased efforts at respiration, or possibly a rupture of the aorta.

Diagnosis: 1. Essential hypertension
2. Intraventricular hemorrhage
3. Aortic aneurysm.

DIAGNOSIS OF THE ACUTE EXANTHEMATA

By D. J. BREITHAUPT, '41

The acute exanthemata are scarlet fever, simple measles, German measles, chickenpox and smallpox. These are the commonest communicable diseases met in children, and are all transmitted as upper respiratory diseases. They are all believed to be due to virus infection with a generalized systemic response, except scarlet fever which is caused by various strains of haemolytic streptococci. The skin reactions seen in these diseases have to be differentiated from allergic responses to serum or drugs, urticaria, and even underwear itch, as well as secondary syphilis and sometimes scabies.

Nomenclature

The latin names may tend to be a bit confusing. Variola is smallpox; varicella, chickenpox. Both start with a "v" as does "vaccination", the prophylactic measure against smallpox. And "varicella" contains the "c" of "chicken-pox"!

Rubeola is used for both simple and German measles. Rubella is reserved for German measles only. As in the above, the "ella" ending denotes the milder disease.

Eruptions

There are four main types.

1. *Erythematous, or scarlatiniform*: This is a general pink blush with local tiny, intensely red spots. It may have a rough feel, simulating "goose pimples" on a blushed skin. The redness may be accentuated by pressing the spread fingers, on the rash to show alternating white and red areas when the hand is removed. An oblique light will tend to show the elevations or punctae.

2. *Morbilliform*: This is a blotchy redness, affecting small irregular areas, with normal skin intervening (not a generalized blush), is palpably elevated with a velvety feel, and tends to coalesce in the more severe stages.

3. *Urticarial:* This is the familiar wheal, with its raised pale center, of variable size, and a surrounding pink hyperaemic areola.

4. *Haemorrhagic:* This is a staining of the tissues by extravascular blood, and the color is not obliterated by pressure, actually a purpura.

The eruptive changes which appear on the skin are known as the exanthem, while the enanthem is the disturbance of the mucous membranes, especially the mouth and throat. A person with a rash should be examined in daylight, the whole body exposed, and the relative distribution of the eruption noted. Is it more peripheral, as in smallpox, or more on the trunk as in chickenpox? Is it mild and discrete as in German measles, or tending to be confluent like simple measles? Does it involve the face definitely, as in simple measles, or is there only a blush on the face with a pallor about the mouth, and an increased rash in the axillae and groins as in scarlet fever? Does it itch, as in scabies and urticaria, or is itch absent as in secondary syphilis. Is there any general constitutional reaction or does the patient feel well? Is there any history of contact with, or previous attacks of communicable disease? Has the patient been taking any drugs, or does he know of any precipitating factor?

Incubation period

The incubation periods may be divided into three approximate classes in which it will be seen that the milder diseases have the longer incubation period.

1. Short, 7 days, Scarlet Fever
2. Medium, 7-14 days, Simple Measles
   Smallpox
3. Long, 14-21 days, German Measles
   Chickenpox.

Invasion or prodromal stage.

This is the period between the onset of first symptoms and the appearance of the true rash. It is characterized by general upset,—malaise, pyrexia, restlessness, chilly sensations, perhaps even rigors, pains in the limbs, nausea and vomiting, and sometimes convulsions, especially in children. These are com-

mon to many of the infectious diseases, and one can say only that the patient has an infection, and not make a diagnosis.

Three diseases, chickenpox, smallpox, and simple measles may occasionally exhibit a prodromal rash, which appears early, is extremely variable in its type, and fades before the true rash appears. It may be morbilliform or erythematous.

The Exanthem

Scarlet Fever

The typical erythematous, or scarlatiniform rash is a collection of closely set minute red points or vesicles, which may be raised like "goose-skin" on a paler flushed background described as being punctate. The skin of the area is entirely involved, i.e. there is no normal skin between the individual red points. The rash appears 3–24 hours after the first symptoms, on the sides of the neck, axillae, upper chest and groins. Later it may involve almost the whole body. There is no true rash on the face, which merely shows a blush, and usually a marked pallor about the lips. On the arms the rash may be blotchy, simulating measles, so that the whole body must always be examined. There may be a pebbly appearance, like water blisters, on the skin of the fingers and toes. Often there is a definite staining of the creases in the skin of the axillae and cubital fossae. This stain is not blanched by pressure. Occasionally one finds petechiae in the anterior folds of the axillae or chest. The eruption may itch.

Simple Measles

The true exanthem may be preceded by a fleeting erythematous or morbilliform prodromal rash. The typical morbilliform measles rash consists of dark red macules or maculo-papules that appear first behind the ears and on the upper forehead. It has a velvety touch, and the whole body is involved in a few hours. At first there is normal skin between the red areas, but the macules tend to coalesce, so that blotchy areas are completely overrun by the coarse dark red eruption. The macules may appear in crops, especially on the face. The circumoral area is always involved, but this may be difficult to recognize in a tanned individual.

German Measles

The rash is similar to simple measles, but less severe, and comes on after 1 day's illness instead of 3–4, and the patient shows very little systemic disturbance. The rash is composed of discrete pink maculo-papules which are all of the same size, but irregular in order of appearance (the simple measles rash appears in an orderly fashion from above downwards). Often the macules are so close together that a scarlatinal rash is simulated. However, the intervening areas of skin are normal, not blushed. The German measles rash is of short duration—1–2 days,—and there is no prodromal rash. In roseola infantum, a rare disease occurring in children under 2 years of age, the rash does not appear until after an abrupt pyrexia, is most evident on the shoulders and back, and fades within 48 hours.

Chickenpox

Within the first 24 hours an erythematous or morbilliform rash may occur, most often seen on the back. The invasion period is very short, and the true rash, at first macular, becomes raised and palpable within a few hours and may itch. In about 24 hours, small, tense, superficial vesicles are formed looking like drops of water on the skin, which may have a slight areola. By the third day pustules have formed, while new crops of vesicles have appeared, so that lesions in all stages,—macule, papule, vesicle, pustule,—are present at one time. By the fifth day scabs have formed over the first lesions. The eruption tends to be centripetal in distribution, i.e. there are relatively more lesions on the trunk than on the limbs.

Smallpox

In practically all the respects mentioned above, the more severe, and now rather rare smallpox differs from chickenpox. The presence of a vaccina-

tion scar caused within the last 7 years is practically positive proof against smallpox. The prodromal rash may simulate scarlet fever or simple measles, be urticarial, or even haemorrhagic. There is a prodromal period of marked prostration for 3–4 days before the true rash appears. This eruption appears first as flat macules, looking like angry flea bites, which in a few hours become papular. Two to three days later, on about the 6th day of the disease vesicles are formed (not on the second day of the disease as in chickenpox). The lesions are all at the same stage of development at any time; new crops are not formed. The eruption is more dense peripherally, on the hands and feet, and above the nose, than on the trunk. This description applies to unmodified smallpox.

A diagram may help contrast these two diseases.

chickenpox	day of disease	smallpox
macule		
papule	1	
vesicle	2	macule
pustule	3	papule
	4	
scab	5	
	6	vesicle
	7	
	8	
	9	
	10	pustule
	11	
	12	
	13	
	14	
	15	
	16	
	17	scab
all stages at once. more on the trunk.		all at same stage on the extremeties

The Enanthem

The conjunctivae tend to be congested, with increased lachrymation in simple measles, rarely in German measles, and are usually clear in scarlet fever.

It is very important that the mouth and pharynx should be inspected. The buccal mucosa may show Koplik's spots, pathognomonic of simple measles. These are minute pin point greyish white specks, like grains of salt, on a red base, usually discrete, but they may be confluent and resemble the appearance of ground glass. They increase for 2–3 days until the true rash appears, then fade.

The tongue is most characteristic in scarlet fever. Early it is coated and whitish, then the papillae desquamate and stand out like the individual parts of a raspberry against a white background. Later, about the fourth day the whole tongue desquamates, giving it a beefy red appearance. By the seventh day the tongue becomes a dull glazed color. In simple measles the tongue may be furred and moist, but otherwise is fairly normal.

The tonsils and pharynx may be quite sore in scarlet fever, simple measles, chickenpox or smallpox. In scarlet fever they are nearly always congested and fiery red, especially when this is the portal of entry, and there is occasionally a white or yellowish tonsillar exudate which is easily rubbed off. This is in contrast to the grey tenacious diphtheritic membrane.

The soft palate in scarlet fever is an evenly stippled red, whereas in simple measles it is a more blotchy dull red, and is only slightly affected in German measles.

In chickenpox and smallpox the typical skin lesions may appear on the buccal and pharyngeal mucosa and become pustular even before the skin eruption is very far advanced. Such lesions are never found in urticaria papulosa or scabies.

Cough: Cough is most often found with simple measles, although German measles may show it to a mild degree.

Glands: Enlargement of the cervical glands is frequent in scarlet fever. With a faint rash, and enlarged glands, especially behind the ear and in the posterior triangle of the neck, German measles is strongly suggested.

Fever: Pyrexia is quite constantly

present. In scarlet fever there is usually a rapid rise to 101–104-F., which is maintained for about 3 days, then gradually falls to normal over a period of 4–6 days. In contrast to this, the onset is irregular in simple measles, often with a few remissions, and there is a rapid fall. German measles shows a slight fever which has disappeared by the third day. After the initial fever with the appearance of the rash, chickenpox may show slight pyrexia with the crops of pustules, while in smallpox the temperature becomes normal about the fifth day, then rises again with the appearance of the pustules, reaching its peak about the tenth day.

Blood: Scarlet fever shows a leukocytosis with neutrophilia and some oesinophilia. Simple and German measles show a more normal count or leukopaenia, with an increased percentage of lymphocytes and monocytes, more marked in simple measles. In smallpox the count falls for 4–5 days, then rises in the pustular stage.

Accessory aids

The Schultz-Carlton reaction of blanching of a scarlet fever rash by the intradermal injection of scarlet fever antitoxin diluted with saline 1:10 to 1:100 may be helpful but is rarely used. The Dick test is positive until after the third week in scarlet fever, and if previously positive, it becomes negative, and is very suggestive evidence that the person had scarlet fever. Desquamation usually occurs in scarlet fever, first on the tongue about the fourth day, then on the cheeks and lobes of the ears by the seventh day, and finally on the hands and feet about the second to third week. It is important to look between the fingers, as callouses will often desquamate with rest alone. There may be some desquamation following simple measles.

Illustrations

Rashes—Harries and Mitman, Clinical practice in Infectious Diseases 1940.
Measles rash—Holt and McIntosh, Diseases of Infancy and Childhood. Ker, Infectious Diseases 1920.
Koplik's spots—Griffith and Mitchell, Diseases of Infancy and Childhood. French, Differential Diagnosis.
Smallpox—Schomberg and Kolmer, Acute Infectious Diseases. Ricketts and Byles, Diagnosis of Smallpox.

To Dr. Beverly Hannah, the author is very indebted for much valuable criticism. My thanks are due also to Dr. H. Jaques for many helpful suggestions.

"The Venous Hum—An Important Clinical Sign!"

"Pressure upon the descending vena cava or the left innominate vein gives rise to a hum, and on the pulmonary artery to a systolic murmur heard best at the second left interspace. But long before the ordinary signs of pressure on the vessels can be detected, we can induce pressure on the vein if the bronchial glands are enlarged. This sign is one of the earliest indications of disease in these glands. Thus, if the child be directed to bend his head backwards upon his shoulders so that his face is turned upwards to the ceiling above him, a venous hum, which varies in intensity according to the size and position of the swollen glands, may be heard with the stethoscope placed upon the upper bone of the sternum. The explanation of this phenomenon appears to be that the retraction of the head tilts forward the lower end of the trachea. This carries with it the glands lying in its bifurcation, and the left innominate vein is compressed where it passes behind the sternum."

A Practical Treatise on Disease in Children,
By Eustace Smith, M.D.
William Wood & Co.
56 & 58 Lafayette Place—1884.

THE OPHTHALMOSCOPE AND THE FUNDUS OF THE EYE

By P. R. BLAHEY, B.A., '41

PART II

(I) THE OPTIC NERVE

1. *Papillitis and optic neuritis:* They are characterized by hyperaemia, inflammatory exudates, oedema and haemorrhages on the optic disc, the margin of which is blurred. The accompanying swelling of the disc fades gradually into the adjacent retina and in general does not exceed two or three diopters in depth. In retro-bulbar optic neuritis the only finding expected is a slight fullness of the vein in some cases. Clinically in both there is a sudden loss of vision with a subsequent total or partial recovery.

2. *Papilloedema:* Papilloedema or a swelling of the optic disc is due to a raised intracranial pressure which obstructs the circulation of the retina. Clinically it is characterized by a gradual and progressive loss of vision.

The optic nerve projects forwards. The affected zone is delimited and localized to the immediate vicinity of the optic disc. It first begins as a passive congestion with fullness of the veins and slight oedema of the disc. Intense oedema of the optic disc may result in a swelling of 8-9 diopters, a condition known as "choked disc". The veins become distended and tortuous and here and there the vessels are hidden by exudates and haemorrhages which occur on the surface of the swollen and distorted disc. Pathological tortuousity of the vessels is noted in that they curve at right angles to the plane of the retina. They tumble as it were over the edge of the swollen disc. The arteries are smaller than normal and are partly obscured by the presence of exudation.

In measuring the extent of swelling of the disc the pupil is dilated and the patient's gaze is fixed on a definite object. A technique described as the "fog" method is used since it ensures the greatest relaxation of the observer's accommodation. The upper temporal vessels are observed with the highest plus lens that will clearly distinguish the borders, then the lower temporal vessel in the same manner. The average of the two readings is taken and subtracted from the number of the plus lens, that will focus clearly on the vessels on the disc. This result gives the number of dioptes of the papilloedema. A swelling of 3 diopters is equivalent to 1 mm.

3. *Optic Atrophy:* Atropy of the optic nerve may be primary (e.g. locomotor ataxy), postneuritic or follow degenerative changes in the retina. When optic atrophy is seen in a late stage, it is difficult to be certain of its pathogenesis. In all cases the disc is pale, excavated, and the small vessels drop over the edge of the retina to sink to a deeper level before they cross the disc.

In atrophy without a previous optic neuritis the thinning of the nerve-fibres renders very visible the fibres of the lamina cribrosa, and the disc acquires a grayish fenestrated or mottled appearance. Atrophy of a post neuritic origin will show local evidence of a previous inflammatory condition. The edge of the disc becomes indistinct and white streaks of fibrous tissue may be seen to pass from the edge of the disc along the vessels into the retina. In cases where a change in the retina precedes optic atrophy, evidence of the former is clearly noted by disturbances of the pigment.

(II) ARTERIAL DISEASE

1. *Atheroma:* This disease of the arteries is characterized by a subendothelial fatty deposit which may later calcify and which is accompanied with a fragmentation of the internal elastic lamina. It affects chiefly the larger branches of the central artery, and is best observed over the area of the optic disc as a narrowing and irregularity of the arterial stream, with a thickening of the vessel wall. When fibrosis supervenes milky streaks become visible adjacent to the blood column. There is a diffuse narrowing of the whole arterial tree. This is attributed to a partial obstruction of the lumen since the athero-

matous change is usually more extensive in the artery upstream. There is an accompanying shrinkage of the artery with a consequent loss of normal tortuosity, the formation of more acute angles at the sight of bifurcation and a displacement of the arterio-venous crossing.

The presence of an atheroma of the retinal vessel occurs in a majority of elderly people. It is a disease which progresses slowly and may be interrupted by vascular accidents. Ocular vascular accidents, such as arterial or venous occlusion and haemorrhages are of no special prognostic value and indicate only that the disease has reached an advanced stage in some particular blood vessel. They, however, warrant an assumption that other vessels are also severely affected (most commonly cerebral arteries) and that other vascular accidents, which may be insignificant or fatal, are likely to occur.

2. *Obliterative endarteritis:* This is a term applied to changes seen in the vessels which nourish atrophic organs. Most characteristically the vessel atrophies and the endothelium proliferates to an extent that it may occlude the vessel. The atrophic lesions of the eye where such findings are observed are glaucoma and optic atrophy.

3. *Arteriolar Sclerosis and Hypertensive Retinitis:* Arteriolosclerosis is characterized by a hyaline degeneration of the media affecting chiefly the smaller arterioles and capillaries. When the disease is generalized, hypertension can often be shown to have preceded opthalmoscopic visible changes in the retinal arterioles. However, the visible vascular changes may indicate the trend of events even before the generalized vascular changes have become obtrusive, or indeed, long before the patient complains of symptoms. The disease may be a unilateral local manifestation in the eye, but is more commonly only a part of a more serious generalized vascular disturbance.

Ophthalmoscopically the larger retinal arteries are usually full and pathologically tortuous. They commonly turn sharply at right angles instead of the normal serpentine tortuosity.

Structural changes in the vessel wall may be observed at the bends. As the disease progresses the arterial wall becomes visible as a glossy coat, the light streak widens and changes are observed in the arterio-venous crossings.

In hypertensive cases transitory local spasms are not uncommon. A permanent constriction is attributed to a thick wall which encroaches on the lumen. In hypertension the retinal arterial pressure is raised, and when this pressure reaches 70 mm. Hg. (140 mm. Hg. systemic diastolic pressure) the force on the capillary bed is so great that it favours oedema and haemorrhages.

Spasms or structural occlusion of the retinal arterioles produces minute focal necrosis in the superficial layers of the retina which appear as greyish fluffy spots. Secondary to the local anoxaemia there is often a generalized capillary hyperaemia but often is specially marked around the fluffy areas of exudate. Petechial and flame shaped haemorrhages are frequent. In a severe form of the disease the characteristic feature is retinal oedema which may be so extensive as to produce a detachment of the retina and precipitate severe haemorrhages.

Most patients with a severe form of retinitis die before the local disease has run its complete course. If they survive long enough, the haemorrhages and exudate disappear and are replaced by sheets of glial and connective tissue fibres. The disc, which is no longer oedematous, becomes pale and atrophic with a blurred margin.

Clinically, arteriolar sclerosis is a disease which in different individuals progresses at a different pace which may change from time to time. A sudden increase in the rate of progress of the disease is often accompanied by new retinal lesions—haemorrhages, exudates and oedema. This justifies an ominous prognosis that few patients will survive more than two years after the appearance of a well-developed retinitis. In cases where the disease is progressive but slow, advanced structural changes in the larger vessels take place without any retinitis. In such instances clinical observations on the condition of the heart

and kidney are more reliable guides for a prognosis.

(III) RETINITIS

A group of diseases in the process of which degenerative or inflammatory changes occur in the retina.

1. *Renal Retinitis (Albuminuric Retinitis):* Retinal changes are rare in acute nephritis. The characteristic form of retinal changes are seen in chronic nephritis while the most severe form is associated with nephritis which accompanies toxemia of pregnancy. Intraocular vascular damages which are associated with nephritis and which usually are accompanied with an elevation of blood pressure may affect a young child and any age adult.

The retinitis is a severe characteristically bilateral retinal toxemia. There is a marked oedema of the nerve head and retina, flame-shaped haemorrhages and gross exudates of a "cotton wool patch" character. The lesions are distributed chiefly in the central part of the fundus. A typical stellate figure is often formed about the macula. This figure is made of globules of exudate along the folds of the internal limiting membrane which is carried forward by the oedema.

The prognosis in the presence of renal retinitis due to chronic nephritis is limited from a few months to the accepted time limit of two years. Should the patient survive long enough that the retinal condition subsides, the optic disc will show variable degrees of secondary optic atrophy, usually some peripheral vascular narrowing, and fine dusty gleaming areas of exudates and a few haemorrhages scattered about the fundus. There may be some areas where the pigment shows a tendency to proliferate.

2. *Retinitis in Toxemia of Pregnancy:* The weight of evidence indicates that the acute late toxemia of pregnancy presents a hypertensive syndrome characterized by a generalized spasm of the small arteries. The degree of systemic vascular spasm appears to parallel the height and duration of the blood pressure and the severity of the toxemia. The change found in the retinal vessels apparently are a measure of the degree of angiospasm throughout the body.

Retinal changes which accompany the toxemia of pregnancy should be added to the accepted diagnostic triad, hypertension, albuminuria and oedema. About one-half of the cases of toxemia show some form of retinitis. For descriptive purposes it may be subdivided into:

1. Oedema of the disc and retina.
2. Cotton wool patches of exudate and areas of haemorrhage.
3. Diffuse retinitis of an albuminuric type.

In the presence of toxemia, ophthalmoscopic examinations are of value in the diagnosis of the severity of the disease, the prognosis and management of the pregnant state. The presence of retinal changes of a comparatively mild degree, or the absence of such changes indicate that pregnancy may be allowed to continue without unduly jeopardizing the safety of the mother and child. It is generally stated that the detection of cotton wool patches of exudate and haemorrhages should always indicate the necessity for an immediate therapeutic abortion. The retintis associated with toxemia of pregnancy subsides rapidly after the termination of pregnancy often with a restoration of good vision.

3. *Diabetic Retinitis:* The retinal changes which accompany diabetes are seen in middle aged and elderly people and rarely in the more severe forms of diabetes mellitis of early life. Associated with it there is commonly some retinal arterial disease. The retinal changes are of no value as an index in reference to the course and prognosis of the disease. Insulin or any other treatment of the diabetes does not have any influence on the retinal changes.

The retinitis is a clinical entity which is attributed to the vascular degeneration and is characterized by small round or rosette haemorrhages and very small sharply defined areas of yellowish white exudate in the central part of each fundus and the absence of any oedema of the optic disc and retina. Individual haemorrhages and patches of exudate may disappear and others appear. Good vision is usually maintained and any decline is likely due to gross intraocular haemorrhages or the presence of a catar-

act. Recently several authors have described the presence of small aneurysmal dilatations of the small retinal blood vessels as a part of the picture of retinitis due to diabetes mellitis. Their significance is not understood.

In the severe forms of juvenile diabetes mellitis, in the presence of a high percentage of fat in the circulating blood, the vessels appear to be filled with a milky fluid. This form of "lipaemia retinalis" is rare and when present indicates a grave metabolic disorder.

4. *Syphilitic Retinitis:* This is seen in congenital syphilis or in the second stage of the acquired form. The inflammation may be almost pure retinal. It is described since other forms of chronic inflammation of the retina may present a similar picture.

Accompanying the low grade inflammation there is a considerable degree of figrosis of the retina and disturbances of the vitreous gel. Syphilitic retinitis is characterized by vitreous opacities, a pale whitish retina, with attenuated vessels sheathed in white fibrous tissue and the presence of optic atrophy. Accompanying this there may be a fine dusty proliferation of the pigment giving a "pepper and salt" fundus.

5. *Pyaemic Retinitis:* Infected emboli usually lodge in the choroidal vessels with secondary involment of the retina. It is described as a white patch with haemorrhages. The appearance of the retina on occlusion of the retinal artery has already been described.

6. *Petinitis Rigmentosa.* Retinitis pigmentosa is defined as a non-inflammatory progressive atrophy of the retinal and choroid, characterized by pigment proliferation and migration of a characteristic type. The typical history of retinitis pigmentosa is a reduction of visual fields to a fixation point which is often well maintained, night blindness, and a long history of defective sight, of which the latter is most constant.

Two conflicting views are held concerning whether retinitis pigmentosa is an abiotrophy of the rods and cones or a result of sclerosis of the choroidal vessels upon which the rods and cones depend so largely for their nutrition. The exact nature of this entity is not understood.

Clinical symptoms precede any evidence of retinitis pigmentosa on ophthalmoscopic examination. Later in the disease, the peripheral part of the retina is studded with characteristic pigmented small spider-shaped spots, which lie in the superficial layers of the retina and tend to aggregate near blood vessels and to migrate peripherally. This migration leaves the pigment epithelium decolorized and consequently a close network of large choroidal vessels becomes clearly visible. The macular region is last to become involved. As a result of retinal degeneration there is an accompanying progressive atrophy of the optic nerve.

The degree of proliferation of the pigment varies widely. In the so-called atypical form of the retinitis pigmentosa the pigmentary change does not occur. The pigment first becomes visible as dark patchy areas due to a thinning of the superficial retinal layers and at a later stage the pigment disappears. It leaves only the white choroid in which there is an extensive sclerosis of the vessels. Ophthalmoscopically it seems justifiable to separate this group from the rest. Likewise, it is differentiated clinically by absence of any family tendency and a probable different etiology.

7. *Choroidal Abiotrophy.* As the name implies, it is by nature a sclerosis of the choroid. Various forms are described with respect to distribution, namely: generalized, central, pericapillary, or a combination of the latter two.

Ophthalmoscopically it may be indistinguishable from the late stage of the so-called atypical retinitis pigmentosa. The area involved presents a network of blood vessels with thick sclerosed walls and a very narrow blood stream. Later stages reveal choroidal sclerosis with complete disappearance of all vessels. The overlying retina atrophies. Pigmentary disturbances are usually a minor part of the picture.

8. *Central Retinal Abiotrophy:* This is a form of macular dystrophy which is accompanied by a gross impairment of vision. This congenital group with a high familiar incidence may occur at any age. It blends with the senile group where heredity is doubtful. Subjectively the macular dystrophies typically lead to

a rapid loss of vision, but it may be slow and the presence of good central vision is not unusual.

The classical concept of macular dystrophy is that of a progressive lesion, more or less confined to the central or perimacular area, which opthalmoscopically shows a protean character. It is diagnosed on the finding of lesions which are described as either one of or a combination of:

1. Fine pigmentary disturbances at the macula.
2. Exudative reactions which are seen as whitish dots, discrete or honeycomb in pattern on a massive area.
3. Atrophic changes seen as a loss of retinal lustre in the macular area.
4. Possibly haemorrhages.

9. *Senile Degeneration of Retina.* Most of the retinal changes which accompany senility are attributed to choroidal vascular changes. The changes are bilateral and symmetrical. The picture presented may be varied degrees of pigment disturbance with a mottled macular area together with atrophy of the retina and choroid. In more severe cases the choroidal vessels are clearly visible. Rarely it takes the form of a macular haemorrhage which may be severe. Clinically central vision is impaired or lost while the peripheral visual field remains normal.

IV VITREOUS DETACHMENT

Only extensive separations become visible. Characteristically the hyaloid membrane assumes an ever-changing wavy form and by reason of its weight, in the absence of adhesions, it will lie in the lower part of the eyeball.

V DETACHMENT OF RETINA

Detachment of the retina is defined as a splitting of the retina whereby the inner layer of cells and fibres become detached from the pigment layer which remains attached to the choroid. The cause of the detachment may be due to inflammation, exudates, haemorrhages, or trauma.

The fundus reflex from the detached area is grayish or darker in colour. The retina is sharply defined and lies in wave-like undulations. A tear or hole is usually seen as a glowing red area in the detached retina. If the macula is detached, it is distinguished as a small fenestrated area.

VI GLAUCOMA

This is a clinical entity which a student learns to fear more than any other catastrophe by reason of the diagnostic uncertainty of the preglaucoma state. Since the essence of glaucoma is an increase in the intraocular pressure, all the tests devised are based on early manifestations as a result of increased pressure. Likewise subjective and objective signs are directly related to the same cause.

Certain tests and findings are:

1. Elevation of eyeball tension. This is determined by palpation or by the use of a tonometer.

2. Pupil reactions: In a normal eye, when a narrow beam of light from a slit lamp is thrown obliquely upon the edge of the pupillary border of the iris; the pupil will alternatively contract and dilate at a rate of 11 to 14 contractions in ten seconds. In the early compensated cases of glaucoma the rate is slower than normal. This altered rate is based on the same factor that causes the well-known tendency of the iris to dilate unduly in glaucoma.

A sluggish oval pupil with the long diameter in the vertical meridian suggests the possibility of increased tension.

3. Central field defect: This signifies hypertension has already been in existence sufficiently long to account for the defect. In a pre-glaucomatous eye an increase in intra-ocular pressure due to the use of a mydriatic may cause a detectable temporary central field defect. In later stages when the hypertension has existed for an appreciable length of time, nerve-fibre degeneration appears and a central scotoma is present. On examining the eye various grades of optic atrophy are noted, depending on the extent and duration of the hypertension.

Summary

The use of the ophthalmoscope and the common findings in the fundus of the

eye are briefly discussed. Where possible an attempt is made to co-relate the findings clinically on a basis of significance, prognosis and treatment.

It is little to say that the fundus of the eye is a picture gallery to which access may be easily and quickly gained and which, like the countenance of the face, varies with each individual, yet a confirmed abnormal finding will clearly indicate the nature and progress of a biological test on living tissue in an environment which cannot be duplicated outside the body. Many of the results of this test, i.e. retinal findings, have in the past been confusing, but with an increase in the use of the ophthalmoscope it has become within the means of everyone to make definite observations which have a clinical significance.

An Inventor of Bones

"How many bones in the human face?
Fourteen, when they're all in place.
How many bones in the human head?
Eight, my child, as I've often said.
How many bones in the human ear?
Four in each, and they help to hear.
How many bones in the human spine?
Twenty-four like a climbing vine.
How many bones the shoulders bind?
Two in each—one before, one behind.
How many bones in the human arm?
In each arm one; two in each forearm.
How many bones in the human wrist?
Eight in each if none are missed.
How many bones in the palm of the hand?
Five in each, with many a band.
How many bones in the fingers ten?
Twenty-eight, and by joints they bend.
How many bones in the human hip?
One in each; like a dish they dip.
How many bones in the human thigh?
One in each and deep they lie.
How many bones in the leg from the knee?
Two in each we can plainly see.
How many bones in the ankle strong?
Seven in each but none are long."

Taken from a Student's notebook.
(Subscribed by Dr. S. M. Hay 8T5)

CARCINOMA OF THE TONGUE

By LIONEL R. DENT, '42

Carcinoma of the tongue is a malignant epithelial growth of the tongue, arising from the stratified squamous epithelium which covers all exposed surfaces of that organ. Certain terms must be identified with the organ for practical purposes. The tongue has two parts,—an anterior two-thirds and a posterior one-third. The anterior two-thirds arises from the floor of the mouth and is called the oral part or body. The posterior one-third forms the anterior wall of the pharynx and is called the pharyngeal part or root. The boundary between the oral and the pharyngeal part consists of a V-shaped line, the sulcus terminalis, extending from each side at the palatoglossal arches to meet in a posterior plane at the foramen caecum in the midline. The dorsum is convex and consists of the superior surface of the oral part and the posterior surface of the pharyngeal part. The apex of the oral part rests against the incisor teeth. Its borders are in free contact with the gums and teeth. The mucous membrane rests on a fibrous stroma, the tunica propria, which covers the dorsum, tip, borders and inferior surface. The surface layer of the mucous membrane is of stratified squamous epithelium, The dorsum is marked with papillae which are minute projections of the corium under the epithelium and these give the tongue its characteristic roughness.

Carcinoma of the tongue makes up 20% of intra-oral tumours. It is a disease of middle or old age, although 25% of cases are under the age of 40 years. It is preponderantly a disease of men, 87% of cases being in the male. Sites of origin in order of frequency are the middle third of the lateral border as in 55% of cases, the pharyngeal part or root in 30% of cases, the dorsum of the oral part in 15% of cases. Points of origin tend to be more common on the border than on the dorsum. This may result from the fact that pre-cancerous lesions are more common here than on the dorsum or pharyngeal part.

Etiology

Carcinoma is said rarely to occur in a normal tongue, that is to say a pre-cancerous lesion has been found to exist in practically all cases. Most pre-cancerous lesions take the form of chronic irritation whether physical, chemical, or pathological in nature. The following types include the more prominent pre-cancerous lesions:

(*a*) *Tobacco:* This is a common form of chronic irritation, but this habit is so widespread one is not justified in making it a definite cause, that is, something may have to be associated with it. Certain experiments have shown that there is no evidence in support of the contention that tobacco smoke contains a cancer-producing property and although a substance was obtained from tobacco which could produce ulcerative lesions, no tumour formation supervened. It seems that some other type of lesion must be associated before a malignant change will occur.

(*b*) *Syphilis:* Many persons with a lingual cancer give a history of syphilitic infection, a positive Wasserman, or show evidence of a syphilitic glossitis. Although the spirochete shows no evidence that it will produce a malignant change, it produces ulceration, scars, fissures and leucoplakia, that predispose to cancer. The primary syphilitic lesions, although a form of chronic irritation themselves, seem to require secondary iniating factors for neoplastic change.

(*c*) *Leucoplakia:* This is essentially a hyperkeratinization and sclerosis of the mucosa of the tongue. It may be local or general, appearing first as raised yellow or white patches on the lingual or oral mucosa. The chief cause of this lesion is also some form of chronic irritation, i.e. oral sepsis, excessive smoking. Leucoplakia in some cases shows a familial tendency and so may explain the early age cancers in some people. It is definitely observed as a pre-cancerous lesion.

(*d*) *Defective Teeth:* Rough and jagged or pointed teeth will quickly pro-

duce a chronic ulcer. It is these chronic ulcers that do not heal in six to eight weeks that tend to a malignant change.

(*c*) *Chemical Irritants:* Certain authorities are of the opinion that strong caustics used in the treatment of chronic ulcers in men over the middle-age group do more damage than good.

Classification

As to morphological types.

(*a*) Ulcerative.
(*b*) Papillary.
(*c*) Nodular.
(*d*) Fissured.
(*e*) Atrophic.

(*a*) *Ulcerative type:* This is the most common type occurring in the oral part, usually taking the form of a non-tender indurated ulcer, arising on the middle third of the lateral border. As it develops the surface usually becomes nodular or granular. The edge of the lesion becomes indurated as the margins of the ulcer become raised and finally the centre becomes excavated. When the growth is 2 or 3 cms. or more in diameter, infection usually occurs and the lesion becomes slightly tender and painful. The edge feels hard on palpation, indurated sometimes like cartilage and the hardness extends into the substance of the tongue to a greater or lesser degree, forming a definite tumour with ill-defined margins.

(*b*) *Papillary type:* This type is highly malignant and highly radio-sensitive. It may vary in size from a small wart to enormous papillary masses filling the entire mouth. In early cases the lesion is circumscribed, soft except at the base and may even be pedunculated. In advanced cases there is an extensive fungating mass, the cauliflower type, with a hard infiltrating base. There may be no ulceration and no bleeding, but once infection has taken place the surface become excoriated and bleeds, the breath is offensive and the growth may be very friable to manipulation.

(*c*) *Nodular type:* This is a less common variety which first appears as a plaque or node, slightly raised above the surface, with smooth epithelium devoid of papillae. It is hard on palpation and usually extends well into the substance of the tongue. Ulceration usually occurs late in the growth.

(*d*) *Fissured type:* Clinically, this variety occurs in the clefts and depressions resulting from old standing syphilitic infection or from chronic superficial glossitis. The fissure is the superficial portion of an ulcerating neoplasm growing in the cleft. Marked induration occurs at the edge of the fissure, and a mass is present deep to it in the substance of the tongue.

(*e*) *Atrophic type:* Also known as the wooden-tongue type. It is a rare form of lingual cancer characterized by slight ulceration, but the tumour produces fibrosis which by contraction gradually shrinks the tongue to an insignificant lump.

The cancer may also occur in a multiple form, usually as two or more distinct epitheliomas which may develop simultaneously having normal epithelium between them. Prognosis in these cases is very poor.

Pathology

Actually there is only one type of carcinoma that can occur in the tongue and that is a squamous-cell carcinoma or an epithelioma, developing from the stratified epithelium of the surface. Histologically the appearance of the disease can be sub-divided into two groups—(*a*) squamous cell carcinoma with cell nests, (*b*) transitional cell carcinoma.

(a) Squamous Cell Type

Columns of epithelial cells, resembling those of the deeper layers of the epidermis and directly continuous with them, grow down into the fibrous tissue and through the fibrous tissue into the muscles. Some columns remain very perpendicular but as a rule they diverge, branch, anastomose with one another and form networks in the deeper layers of the tongue. These downgrowing cells are easily recognized as epithelial, yet they differ in many respects from the normal surface epithelium. They vary in size, usually smaller than the cells of the superficial layers of the epidermis. They vary as much in shape, but are seldom distinctly spindle

or caudate; polygonal cells are very common. Most cells have nuclei that are very large and more deeply-staining than the normal. Multinucleated cells may also be found.

Amongst the larger columns are seen "cell nests" or "epithelial pearls"; these are rounded or oval bodies consisting of one, two or more central altered epithelial cells surrounded by layers of flattened scale-like cells. They are portions of the downgrowing columns undergoing degenerate corneous activity. There is no limiting membrane of any kind, but the neoplastic columns are almost always clearly separated from the surrounding fibrous or muscle tissue.

(b) Transitional Cell Carcinoma

Most characteristic feature of this type is the absence of cell nests. They were first recognized by their marked degree of radio-sensitivity. It is most common on the pharyngeal part of the tongue although it is also found in other parts of the mouth. It is subdivided into two types.

(1) Lympho-epithelioma of Regaud.
(2) Transitional cell type.

(1) *Lympho-epithelioma of Regaud:* Histologically consists of large cells with a delicate cell membrane and trabeculated protoplasm with large nuclei that do not form cell nests. Masses of epithelial cells grow in wide sheets or cords, infiltrating the surrounding lymphoid tissue. Ewing in a review of 300 tumours occurring in this location found 6.3% of cases in this group.

(2) *Transitional cell type:* This type consists of small round cells, some polyhedral, with large nuclei. As a rule little intracellular tissue is present. Squamous characters of the cells are lacking and the cells are not differentiated. These tumours may exhibit a marked degree of anaplasia. The most important clinical point is their marked degree of radio-sensitivity. The rapid retrogression and total disappearance of the primary lesion and temporary disappearance of cervical metastases after a mild radiation was the first feature that attracted attention to these tumours.

Treatment

Treatment is resolved into that of two types.

(1) Treatment of the primary lesion in the mouth.
(2) Treatment of cervical metastases.

(1) *Treatment of the primary lesion in the mouth:* General hygienic measures. Mouthwashes and oral irrigations should be done regularly. Extraction of teeth is advised as a routine measure in some clinics, whereas other authorities are against any wholesale extraction.

Radiation is the generally accepted method of treatment for the primary lesion. Surgical treatment in many cases is difficult and dangerous, due to the fact that many growths arise in inaccessible parts of the mouth and invasion of surrounding structures may have occurred. If a small growth arises on the tip of the tongue or fungates from one of its lateral free borders, surgical removal may often be accomplished with a fair chance of local cure but ordinarily the method is much less conservative and less successful than radiation. Conservation of tissue is extremely important and is more liable to follow radio-therapy than surgery. Due to the anatomical position of some pharyngeal tumours, radiation supplies the only form of consistent therapy.

Radiation of the primary lesion is ordinarily best administered by a combination of external radiation and interstitial radiation. For cancer of the oral part, external radiation perorally may be given advantageously by the use of a metal cylinder of the proper size, so as to limit the diameter of the beam and to insure its proper direction. For pharyngeal cancer external radiation is given through the skin of the neck on each side by the daily fractionated method and supplemented by radon needles inserted through the skin of the supra-hyoid region through the midline anteriorly. Radiation alone is often used to deal the knockout blow to the tumour. If the lesion does not disappear entirely then treatment is

supplemented by the use of interstitial radiation. Following this if part of the lesion still remains then surgical diathermy is used as a last form of treatment. In some cases radon needles may be used alone, these being inserted through the lingual surface into and about the growth. These needles are difficult to keep in position and infection may be introduced through their use.

In any case the appearance of a local reaction to the needles must be noted. Treatment is as a rule well tolerated, pain may be present and 24 hours after the needle insertion the tongue tends to become very edematous. The degree of the edema varies with the condition of the patient and indicates, if severe, a grave prognosis. There may tend to be superficial ulceration for several months after the treatment and this must be distinguished from a local recurrence, which may cause further treatment and aggravation of the condition. Radionecrosis from excessive radiation is a formidable complication. The patient in some cases becomes exhausted from insomnia, toxemia and semi-starvation due to inability to swallow food.

(2) *Treatment of cervical metastases:* At this point a slight review of the lymphatic drainage of the tongue might be advisable. The main chain of glands in the head and neck is called the deep cervical trunk running along the course of the internal jugular vein. Most of this trunk may be placed behind the sternomastoid but several glands may be in the anterior triangle and several in the posterior triangle. Two glands are to be noted; the jugulo-digastric at the posterior belly of the digastric where it crosses the internal jugular, and the jugulo-omohyoid where the posterior belly of the omo-hyoid crosses the vein. The lymph vessels of the tongue ultimately arrive at the glands situated between the jugulo-digastric and the jugulo-omohyoid. Those from the tip pierce the mylohyoid and end up at the submental gland. Those from the submucous plexus of the dorsum pass laterally to pierce the mylohyoid muscle and reach the submandibular glands. The vessels from the pharyngeal part pass through the lateral wall of the pharynx below the tonsil and reach the upper deep cervical glands.

Involvement from the tongue is likely to be bilateral or to opposite side from any portion of the tongue. Surgical dissection of the exposed lymphatic area or radiation may be employed, the case in point will determine the best treatment. Surgical dissection has always the operable risk associated and must be borne in mind. It is associated with a definite mortality and should only be done in consonant with the following conditions.

(1) The primary lesion in the mouth must be entirely healed.
(2) Glands although palpable and clinically malignant, must be operable.
(3) The patient must be a good surgical risk.

Irradiation is done where a block dissection is impossible. The two treatments may be combined together for good results. After a thirty-day interval from radiation of a lingual cancer, a block dissection may be done on the neck, followed in two or three weeks by a course in X-ray therapy to the site of the operation. In the cases where no palpable metastases are found, treatment varies in different clinics. There is the objection to neck dissection because of the operable risk. There is the drawback to caution radiation that the necessary lethal dose is excessive for average tolerance and is therefore unadvisable.

Prognosis

In early cases of operable cancer a 55% cure is expected. The prognosis is much better for growths occurring on the oral part of the tongue. Growths on the pharyngeal part show a poor average prognosis due to the fact that they are discovered later as a rule. The presence of metastases is also indicative of a further stage in the disease and a poorer prognosis.

Following the clinical classification used in this school the associated prognosis is as follows:

Stage 1. A lesion which is 1.5 cms. in

diameter. Prognosis: a five year cure, in 90% of cases.

Stage 2. A more extensive lesion spreading on the surface and approximating 3 cms. in diameter. Prognosis of 70% cure as above.

Stage 3. A lesion involving the greater part of one-half of the tongue and in some cases nearby structures. Prognosis of 40% cure as above.

Stage 4. A lesion with complete involvement of the surface. The prognosis is extremely poor and it could actually be said to be incurable.

It is remarkable the amount of neglect present in some cases when it is considered that the mouth is one of the cavities of the body most open to inspection and that cancers of the tongue are usually preceded by pre-cancerous lesions. Until the public is educated regarding the importance of mouth hygiene and the importance of obtaining medical advice promptly when unusual conditions develop and persist about the mouth and tongue, the percentage cure will continue to remain low.

ANSWER TO PROBLEM IN LAST ISSUE

By F. THOMPSON, B.A., M.D.
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Autopsy Report

1. An aneurism was found at the junction of the thoracic and abdominal aorta lying in front of the ninth dorsal to first lumbar vertebral bodies. The aneurism was thick walled and densely adherent to adjacent structures. There was a saccular projection just above the diaphragm on the right side, this was full of laminated thrombus and communicated with the right pleural cavity by a small perforation. The wall of the aneurism was largely granulation tissue and was densely infiltrated with neutophils as was the thrombus.

2. The anterior surfaces of the ninth dorsal to first lumbar vertebral bodies were eroded, the inter vertebral discs remained normal.

3. The right lung was partially collapsed and there were fifteen hundred cubic centimetres of fluid and clotted blood in the right pleural cavity.

4. Section of the brain revealed bilateral haemorrhage into the subcortical area of the occipital lobes of the brain near the posterior poles. This was more extensive on the right side. No embolus or other finding was present to explain the bilateral nature of these haemorrhages. There was only slight atherosclerosis of the cerebral vessels.

5. Marked adhesions of the left pleural cavity.

Diagnosis

1. Mycotic aneurism at junction of thoracic and abdominal aorta with rupture into right pleural cavity.

2. Bilateral intracerebral haemorrhage occipital lobes.

Discussion

We know from the history and from the presence of dense adhesions in the left pleural cavity that the patient had an empyema which did not heal until June, 1940. This must have been the source of the infection which caused the development of the myeotic aneurism and erosion of the vertebral bodies with the resultant pain in this region starting as it did in July, 1940.

The headache followed by flashes of light and decreased vision were caused by the haemorrhage into the occipital lobes of the brain.

The terminal event was of course due to rupture of the mycotic aneurism into the right pleural cavity causing increased back pain, shoulder tip pain due to irritation of the diaphragm, and marked dyspnoea due to blood loss.

STAPHYLOCOCCAL FOOD POISONING

By R. J. WILSON, M.A., '42

Introduction

One of the most common types of food poisoning is that which has been variously described under the headings of "ptomaine poisoning", "summer complaint", "something I ate" and "housemaid's complaint". It is characterized by the sudden onset of nausea and vomiting accompanied by vertigo and abdominal cramps, and followed by profuse diarrhoea and a varied degree of prostration. Recovery is usually complete within 24-72 hours. The signs and symptoms are familiar even to the public, but little else is generally known about the condition. Within the past decade, the etiology and epidemiology of this type of food poisoning have been investigated by a number of workers in the United States and in Canada and a brief summary of their findings is presented.

In 1914 an outbreak of food poisoning with the symptomatology described above was investigated by Barber in the Philippines (1). It was traced to milk containing large numbers of staphylococci, and the source of contamination was shown to be a cow suffering from staphylococcal mastitis. Barber inoculated sterile milk with the micro-organisms cultured from the lesion, incubated it for several hours and drank a small quantity. He reproduced in himself the signs and symptoms exhibited by those persons involved in the outbreak.

Little notice was taken of Barber's report until 1930 when workers in the United States of America investigated several large outbreaks of a similar type of food poisoning and isolated from the incriminated food large numbers of staphylococci(2). Filtrates of the strains isolated from the foods, when ingested by human volunteers, produced the same acute symptoms as those observed in the outbreaks. Monkeys were used as experimental animals with variable success in the study of this gastro-intestinal poison, but all other laboratory animals were refractory. It was concluded that the poisoning was produced by a soluble exotoxin of the staphylococcus which was termed an "enterotoxin".

In 1936 a group of Canadian investigators(3) demonstrated the fact that the intraperitoneal injection of suitably prepared filtrates of certain strains of staphylococci in kittens produced a syndrome similar in most respects to that observed in humans suffering from staphylococcal food poisoning. Further academic investigation was thereby facilitated and considerable data have been accumulated with regard to the nature of the outbreaks and the properties and mode of action of staphylococcal enterotoxin.

Symptomatology

The signs and symptoms are remarkably constant in the reports in the literature and vary only in degree. The onset is rapid and occurs from $1\frac{1}{2}$ to 5 hours after ingestion of the contaminated food. It is marked by dizziness, nausea and abdominal cramps followed by a projectile type of vomiting which may last only a few hours or, in severe cases, for several days. The vomitus may be blood-streaked. The temperature is usually normal or subnormal in the acute stage. The pulse rate is noticeably increased and there are frequently cold sweats. Diarrhoea may begin with the vomiting or be delayed for several hours and the patients may become markedly dehydrated. The more severe cases are definitely in a state of shock. In a high percentage of instances there is marked abdominal rigidity. The acute symptoms may last only a few hours but because of prostration, recovery may be delayed for several days. During this period a temperature of 100° is not uncommon.

Denison(4), in his investigations, fed sterile broth filtrates of strains with a food-poisoning history to human volunteers. In addition to the signs and symptoms recorded above, which were confirmed, he noted a marked fall in blood pressure and an increase in the

white blood cell count to 15–21,000 with 80 per cent polymorphonuclar leucocytes.

Differential Diagnosis

Since there have been no fatalities recorded in the literature, the chief problem confronting the physician is that of differential diagnosis. While it is true that the only reliable method of distinguishing this type of food poisoning from those due to the Salmonella group is that of bacteriological investigation, it must be noted that in staphylococcal food poisoning the onset is extremely acute in most instances and occurs within $1\frac{1}{2}$ to 5 hours following ingestion of the contaminated food. In poisoning due to the Salmonella group the onset is usually delayed for 12 hours or more. Provision should always be made, however, for bacteriological examination of blood, stools, vomitus and suspected foods. In not a few instances the state of shock, the high white cell count and the rigid abdomen have led to the diagnosis of an acute abdomen, and a laparotomy has been performed. This error can obviously not be avoided in some instances, but it may be again pointed out that in staphylococcal food poisoning the temperature remains normal or even subnormal until the acute stage has passed, and even then it seldom rises to above 100°F.

Treatment

The treatment of staphylococcal food poisoning is symptomatic, and in severe cases is essentially the treatment of shock and dehydration. Bayliss found experimentally in cats that morphine was the drug of choice in reducing the severity of emesis produced by the enterotoxin, while ergotoxine was second. Atropine and pentabarbital had no demonstrable inhibiting effect.

Incidence

In the earlier investigations there was a definite geographic incidence centred chiefly about the southern United States. But when interest in the problem became more widespread, outbreaks were reported in Canada, England, Holland, Germany and Italy.

It is undoubtedly true that outbreaks occur more frequently in the summer months, but this is not invariably the case. Age and sex have seemingly little bearing upon susceptibility to the enterotoxin. Outbreaks most frequently occur in institutions such as boarding schools, hospitals, army barracks, hotels, or at conventions, picnics, church socials, etc., where food is prepared in large quantities and eaten by a number of persons who subsequently become ill more or less at the same time. This serves to excite sufficient suspicion to warrant an investigation. It has recently come to our attention, however, that there are many instances of poisoning in one or two individuals in a family. Recovery is so rapid that an investigation is not undertaken by the physician if one is called. It is probable that the majority of cases are never reported, and it is undoubtedly true that the majority are never investigated. There are few persons who at one time or another have not experienced this type of illness in either mild or severe form.

Almost every common type of foodstuff has been implicated in staphylococcal food poisoning outbreaks at one time or another, but predominant among these are milk and milk products including ice cream, and cream or custard pastries. Over 80 per cent of the outbreaks reported in the literature have been attributed to this group. The remaining 15–20 per cent includes meats, fish, gravies, prepared meats, salads, sandwiches and oysters.

The Enterotoxin

The enterotoxin is a soluble exotoxin of the staphylococcus, and therefore produces essentially a "food intoxication". Its properties have been investigated by Jordan and his colleagues(2) who used human volunteers and monkeys as test animals and by Dolman and Wilson(5) who utilized the kitten as a test animal. They showed that the toxin is relatively heat stable, and can resist a temperature of 100-C. for at least 20–30 minutes. The importance of the heat stability lies in the fact that

the enterotoxin formed in foods before cooking may withstand the process even though the bacteria are killed. This stands in marked contrast to the toxin of *B. botulinus* which is destroyed by exposure to a temperature of 83-C. for 6 minutes, and to the bacteria of the Salmonella group which are destroyed by the pasteurization process. The enterotoxin is antigenic when injected into experimental animals, and Dolman and Wilson were able to demonstrate active immunity in kittens, and the serum of these immune animals conferred passive immunity. They have also prepared specific antitoxic sera in rabbits and in horses.

The mode of action of the enterotoxin has been investigated recently by Milward Bayliss(6) who used cats as test animals. His experimental findings indicate that the toxin is neurotropic, and acts on the peripheral sensory structures of the small intestine. The impulses pass to the vomiting centre mainly by way of the vagus nerves. The efferent pathways are principally directed along the phrenic nerves to the diaphragm, the somatic nerves to the trunk musculature, and the vagus and sympathetic nerves to the enteron. It is of interest to note that in his experiments morphine inhibited, ergotoxine inhibited or delayed, while atropine and pentabarbital had little or no effect on emesis produced by staphylococcal enterotoxin.

Sources and Methods of Food Contamination

The frequent implication of milk and milk products has thrown suspicion upon strains of staphylococci isolated from unpasteurized milk. In a series of surveys by several investigators it was shown that about 40–45 per cent of strains isolated from raw milk and milk from cows known to be suffering from mastitis were able to produce enterotoxin. Several outbreaks have been recorded in the literature as having resulted from the consumption of unpasteurized milk and milk from infected cows, but there remained a large group of outbreaks where this method of infection was obviously not operative. recent investigations of several outbreaks in Hamilton, Canada,(7) involving some 125 persons revealed that the micro-orgamisms which contaminated the food originated in the nose and throat of those preparing it. When one comes to consider the high incidence of staphylococci in the nose and throat of the general population it is not at all surprising that this should be an important source of contamination. Hallman, Hart and McFarlan give the incidence of staphylococci in the nose of the general population as about 76, 70 and 66 per cent respectively.

Another interesting observation was made during the investigation of the Hamilton outbreaks. The same bakery was implicated on two separate occasions within 6 to 8 weeks. The strain isolated from the food in each instance was shown to have come from the nose of the proprietor of the bakery, and further investigation demonstrated the fact that he harboured the identical strain in his nose for a period of over two years following the outbreaks. We have two other cases on record where the same strain was isolated from the nose over periods of several months and 4 years respectively. In none of these cases was there clinical evidence of an active infection. While this concept of "carriers" is of relatively little significance as far as the general population is concerned, it is of utmost importance in food handlers. The nose and throat is, in the author's opinion, the most important source of contamination of foods by staphylococci. While superficial lesions on the hands and face have been suggested as potential hazards, proof of their implication is lacking in the literature. They are certainly the most obvious source to the eye, and must be considered.

Methods of Investigation

The investigation of this type of food poisoning follows routine epidemiological principles. Prompt and accurate case histories are most essential in order to determine the food stuff involved and to obtain adequate samples. If the out-

break is traced to a large bakery or the kitchen of an institution, samples of the ingredients should also be included. These must be promptly submitted to the laboratory in clean, individual containers which should be packed in ice if they are to be in transit for any length of time. Details of the mode of preparation and storage of the food involved should be accurately outlined and submitted with the case histories.

In attempting to ascertain the source of contamination, the nature and source of the milk supply must be determined and adequate samples obtained. Nose and throat swabs of those employees who prepared or handled the food are of utmost importance, and superficial lesions must not be overlooked.

There is little need to enlarge upon the methods adopted by the laboratory(8) in investigating these outbreaks. Toxins are prepared from the strains isolated from the foods, suitably treated, and injected intraperitoneally into kittens. The enterotoxin, if present, produces the typical signs and symptoms of vertigo, nausea (shown by marked discomfort and salivation), followed by vomiting and diarrhoea as described above in human intoxication.

Epidemiology

It is not proposed within the scope of this paper to deal with the problem by discussing it from an epidemiological standpoint, for this is adequately reviewed in the reports of the outbreaks in Hamilton in 1938 and 1939 (7). However, a few words might be included with regard to the control of this type of food poisoning. The source of contamination obviously cannot be controlled for the incidence of staphylococci in the nose and throat provides a well of infection which cannot be readily overcome. Control must be directed along two main channels, namely, improved methods of preparing and handling the foods to minimize contamination, and prompt and adequate refrigeration of the finished products to prevent proliferation of those microorganisms which have gained access to the food. In certain parts of the United States, regulations have been established to cover these and other points (9), and the public are exhorted not to transport or keep these products unless refrigeration facilities are available. Indeed, in some localities where outbreaks have been a frequent source of concern to public health authorities the sale of cream or custard-filled bakery goods is forbidden in the summer months, and in these several ways the incidence of staphylococcal food poisoning has been markedly decreased in the areas concerned. But one feels that a great deal of public enlightenment will be required to persuade Canadian bakers not to display the pride of their trade in warm display windows and cabinets, and to dissuade housewives from buying wares so displayed to tempt the appetites of their families.

References

(1) Barber, M.A.: Philippine J. Sc. 9:515, 1914.
(2) Jordan, E. O.: J. A. M. A. 97:1704, 1931.
Dack, G. M.; Cary, W. E.; Woolpert, O. and Wiggers, H.: J. Prev. Med. 4:167, 1930.
Jordan, E. O.; Dack, G. M.; and Woolpert, O.: J. Prev. Med. 5:391, 1931.
(3) Dolman, C. E.; Wilson, R. J.; and Cockcroft, W. H.: Can. Pub. H. J. 27:489, 1936.
(4) Denison, G. A.: Am. J.P.H. 26:1168, 1936.
(5) Dolman, C. E.; and Wilson, R. J.: J. Immunol. 35:13, 1938.
(6) Bayliss, Milward: J. Exp. Med. 72:669, 1940.
(7) Roberts, James; Deadman, W. J.; and Wilson, R. J.: Can. Pub. H. J. 29:325, 1938.
Roberts, James; and Wilson, R. J.: Can. Pub. H. J. 30:590, 1939.
(8) Wilson, R. J.: Can. Pub. H. J. 31:607, 1940.
(9) U.S. Public Health Reports, June 11, 1937, p. 765.

Feature Section

ALAN VAUGHAN, Editor

A CRITICISM OF HISTORY WRITING ON WARD "A" TORONTO GENERAL HOSPITAL

By MARY V. JOHNSTONE

Secretary

Recently some one asked me to define my conception of Utopia, to which I promptly replied, "a state where no dictaphone exists". Lest I incur the displeasure of the Dictaphone Corporation, let me hasten to add that my experience with this invaluable instrument has been a real challenge to any secretary. I am fully aware of its importance in the economic scheme in many spheres, especially for the secretary with no knowledge of shorthand and no desire to acquire such and to the busy executive who cannot find time to dictate to his secretary during the hours her services are available. But like many of the good things of earth, it is frequently abused.

My quarrel is not with this innocent device but rather with its misusers. My criticism is of history writing by the Sixth year medical students on the surgical wards of this hospital. The capacity in which I function is that of a secretary. For thirty weeks of each year, divided into ten week periods, final year students swoop down upon us and are assigned the task of writing histories; from five to ten students in each group, varying according to the extent of the final year group. They take histories and examine the patients, proceed to dictate their findings to the dictaphone, to be transcribed by the secretary.

The time these students are thus engaged is too limited to permit them to become proficient with the use of the machine. There is, of course, the exceptionally bright lad, who right off the bat, knows how to handle it, taking to it like a duck to water and causes one little concern with the transcription. These fellows are as rare as the axiomatic "hen's teeth". Usually some time elapses before he gets control; then the vital problem arises.

Picture him, comfortably seated in an armchair, feet on the desk, cigarette in one hand, or more frequently a pipe (perhaps they consider a pipe more professional, judging by the number who seem to prefer a pipe to cigarettes); in the other hand he holds the formidable trumpet to his mouth, his inhibitions all removed and nothing to stop him pouring out his soul except his conscience and the anticipation of the secretary's wrath on receipt of the results. There is the tendency to ramble aimlessly on enumerating all sorts of irrelevant findings on physical examination. Here the student should realize his opportunity to learn to write histories, such as he will keep when he graduates to private practice. It is the universal experience that if he does not keep such histories, he degenerates rapidly and his patients will suffer. In private practice he will, in all probability, use a form history. The space for the history will be small. It is therefore of great importance that the recording of the history be concise and telegraphic. That is, no useful word must be left out and no excess word inserted. All such phrases as "patient states" are so much wasted ink and energy.

Space will not permit me to record one of these detailed histories, but let me quote from some of the records.

The chief offender is the verbose fellow. He leaves no stone unturned. He supplies an amazing amount of information, economic, personal and spiritual. He

goes in to the most minute details; how much rent the patient pays; if purchasing his home, the amount of mortgage he is carrying; the ages of the various members of the immediate family and all the facts he can glean about his ancestry as far back as the family tree can be traced; a graphic description of the patient, so perfect, that never having seen him, you instantly recognize him on the Ward by his "big blue eyes". He is very particular regarding the geographic position of the patient in bed, whether he is sitting up or huddled under the covers. He never omits to describe in detail the dandruff with which the patient is afflicted—great flakes, resembling sugar. A tattoo mark is another subject for lengthy discourse. "The patient's left arm presents a tattoo mark. Here is depicted a beautiful maiden playing upon a harp. In the background cupid is represented about to shoot an arrow from his quiver".

Then there is the one who stresses certain subjects to the exclusion of all others of major importance. He unearths a divorce in the family in his questioning, probably a remote cousin. Such artistry does he display in the set up that one is convinced that here is a very essential piece of information.

From a mass of detail in the present illness of a patient on whom a diagnosis of varicose veins was made, one relates the following:—"this patient has a friend who lives in Peterborough. She goes to visit this friend sometimes". There he leaves you to conjecture what you may about Peterborough. No doubt Peterborough is a very delightful spot to visit but as the historian does not specify that the patient walks to Peterborough, one questions the significance of this statement in this particular history of a case of varicose veins.

Repetition is another annoyance in transcribing these lengthy surgical histories; The reiteration of certain pathological findings, describing them finally in a long wordy summary, even though they are not the major condition necessitating the patient's admission to hospital.

I do not wish to minimize the importance of complete investigation but it has been my experience as a medical secretary that too many students fail to see the woods for the trees.

It should be a point of pride with every student, that his histories are clear and concise in order that the staff surgeon in attendance on the case may see at a glance that all the important findings are noted on the chart without reading numerous typewritten pages of non-essential findings. In future survey work it facilitates the drudgery necessary in this particular branch, to work on well written histories and one thus engaged feels the material is reliable and noteworthy.

The writer is fully aware that criticism from a lay person will not be received with magnanimity. Should it produce no other effect than to focus attention on history writing, the effort and the criticism has been well worth while and it may be that future secretaries will consider history writing a pleasant part of their daily routine.

CANADIAN INTERN BOARD REPORT FOR 1940-41

By C. J. M. HEALY, '41
Secretary

Following the sound principles, laid down last year by Dr. William Kerr in conjunction with the Canadian Medical Association and the Canadian Hospital Council, the Canadian Intern Board has completed a very successful second year. Like all new ideas, however, this plan has had to be tried and tested in every possible manner before its true value could be correctly estimated. We have been quite satisfied with the final results, since the board has been placed through

practically every "acid test" in its early infancy and still has every indication of becoming a robust child. However, in presenting this report we want you to be the judges, and any criticism or suggestions for the future will be most welcome. This year a few necessary changes were made. One very satisfactory plan devised this year, was the one whereby the Board submitted to all the hospitals a list of students who were applying through us. This did away with the confusion encountered last year, when a few hospitals placed the names of students on their hospital forms, who had not submitted an application to the C.I.B.

The number of students applying through the Board has almost doubled this year. The following is a summary of the various universities:

University of Alberta	36 forms
Queen s University	26 forms
University of Toronto	115 forms
University of Western Ontario	23 forms
Dalhousie University	1 form
McGill University	1 form
Yale University	1 form
	203 forms

We were particularly gratified by the very fine registration and by the enthusiasm shown by the University of Alberta students.

One hospital remarked that until every student applied, the Board could not operate satisfactorily. For this reason we canvassed the schools to try and determine why we had not received an application from every final year student. The following reasons were given:

1. A certain number of students did not apply to Canadian hospitals and consequently they would not benefit by submitting a form to the C.I.B.

2. A certain number of students at one school applied to a local teaching hospital which did not deem it advisable to submit a list to us this year, due to local circumstances. However, these students did not apply to any other hospitals, so that this did not hinder the functioning of the Board. May we add that this hospital in question has voiced its approval of the C.I.B. and hopes to enter under the scheme when possible?

3. A handful of students at one school was misguided into believing that they had a better chance of obtaining their appointment independently of the C.I.B. at a certain hospital. However, this proved erroneous, since we received the fullest cooperation from that particular hospital in question.

4. At Dalhousie, Laval, University of Montreal and the University of Manitoba the final year students serve their junior rotating internships during their last year at school. Since these appointments are arranged at these centres by selected hospitals and the medical schools concerned, the appointment problem is distinctly a local one. A somewhat similar arrangement exists at McGill.

It may be concluded, therefore, that all eligible students across Canada, with the very rare exception, applied for their internships through the C.I.B. The various representatives at each school have informed us that the plan was very satisfactory this year, as judged from student opinion.

Number of applications received from students	203
Number of applications filled by the C.I.B.	188
Number of applications not filled:	
Alberta 2, Queen's 2, Toronto 10, Western 1	15

Therefore the C.I.B. placed 93%of its applicants. At the present time there are only four of the original fifteen who have still not been placed. We believe that by furnishing each of the above fifteen students with a list of hospitals that did not receive their full quota and that by furnishing the latter with a list of unplaced students, the C.I.B. has made the above possible, to a large extent.

Also this year we have found that the number of broken contracts and "jumping from one hospital to another" has decreased considerably. We have heard of a few unavoidable instances where a student, due to financial or other reasons, was unable to accept his appointment, but in most instances, we were able to have him arrange for a satisfactory substitute to take his place from among those not placed. In cases where a substitute was not available, we did not encourage the move.

The following is a summary of how the students fared in their choice of hospitals:

	Hospital of 1st choice	2nd	3rd	4th	5th	6th	
Alberta . . .	30	2	2	0	0	0	34
Queen's . . .	18	4	1	0	0	1	24
Toronto . . .	79	11	10	2	3	0	105
Western . . .	21	1	0	0	0	0	22
Others . . .	3	0	0	0	0	0	3
Total . . .	151 (80%)	18 (10%)	13 (7%)	4, 5, & 6 (3%)			188 (100%

Occasionally we have heard a student remark that he considers the C.I.B. unnecessary and that he should be able to receive an appointment by means of the old system just as easily.

However, from the above figures and from the data we have at hand, we can definitely state that the 80% who were appointed to their first choice hospital would not all have fared so under the old regime. In fact, we can practically state that instead of 80%, it would have been approximately 55% at the most. It is possible that the C.I.B. does not play as important a role for the honor students, since they would have no trouble in obtaining their appointments, but for the remainder who make up the majority, there is no doubt now that they were benefited.

The only criticism received, with regards to the students' part, this year was that in a half a dozen cases, they failed to apply *personally* to *all* the hospitals they had listed on their forms to us. However, where this was discovered, it was rectified in every case.

Let us now consider how the hospitals fared. We received lists from thirty-three hospitals approved for internship. Hospitals in centres like Montreal, Winnipeg, etc., did not hand in lists, since they obtain their interns from McGill and the University of Manitoba, etc., respectively.

One interesting fact noted was that the sum total of interns requested by the above thirty-three hospitals was 246. Since there were only 203 students applying through the C.I.B., it can be seen that it would have been impossible for us to fill every hospital's quota of interns.

Number of hospitals with quota filled	13
Number of hospitals with quota *not* filled	20

Of the twenty hospitals who did not receive their full quota, many were short one or two men and we understand that they will be able to rectify their loss by obtaining men from the U.S.A., final year students and from students not placed

as yet. Some have already filled their quota by means of the list sent them of unplaced students.

There were, however, five or six hospitals that were left "out in the cold." Of this number we were unable to help in three of the instances. However, in the remaining three cases we found that it was the hospital that was at fault. They were late in forwarding their forms to the C.I.B. in the first place and, in the second place, their lists were not adequate. In one case, all we received were the names of four men with no indication of which were preferred or which were alternate choices. In cases like this, we usually informed the hospital of their mistake and asked them to give us the names of more men, if possible. But in this particular instance, the forms did not reach us until December 12th, about two weeks late, and we were unable to act on such short notice.

The C.I.B. is repeatedly endeavouring to impress upon the student the importance of acknowledging his appointment to the hospital after the final allotment. In a few instances they have failed to do so, but at the time of writing, all have been acknowledged as far as we know.

Perhaps the most serious problem that the C.I.B. faced this year was that several of the hospitals announced their appointments and required those appointed to sign contracts before the announcement of the allocation by the C.I.B. In two or three instances, however, we were assured that it was due to a misunderstanding on the hospital s part and that it would not happen again next year. However, we are afraid that this was not so in every case. May we say that there is absolutely nothing gained by this procedure, since, if a student receives an appointment through the C.I.B. later which he would rather have, as shown on his preference list, he usually decides to break his original contract. Once a student finds out that a certain hospital is breaking the rules, he will have few scruples about breaking his contract if he receives a better offer. This was one of the main drawbacks with the old system and hospitals that do this are just defeating the purpose of the Board. However, we do believe that next year this will be all cleared up.

Likewise one deprecates the practice of certain hospitals forcing the students to divulge their order of preference. In nine cases out of ten the student will probably mislead the examiner, since he realizes that advantage is being taken of him. Those hospitals that practised this lost more than they gained because they were so often mislead by the student's answer.

In conclusion, the C.I.B. has placed 188 interns in Canadian hospitals for the coming year directly and eleven more indirectly. It will endeavour to fill the quota of more hospitals next year, but from the above it can be seen that unless some of the larger hospitals reduce their quota, the C.I.B. will not be able to fill every hospital's requirements. However, we are sure that this shortage would have been just as great, and possibly greater, with the old system and the confusion would have continued until Easter. As it is, all the appointments were made by December fifteenth, the students intend to fulfill their contracts and will not start to "bargain and shuffle" for another hospital in May or June, as so often happened in years gone by. Space has not permitted the presentation of an outline of the method by which the Board functions in this article, but if there is anyone who is not acquainted with these facts, kindly let us know and we will gladly forward the information.

The secretary desires to express his gratitude to Dr. Harvey Agnew, Dr. Fred A. Logan and Dr. William Kerr for their generous assistance and sound advice. Without their help the Board could not have carried on.

To Alumni Notes

By K. C. GREER, M.D.

The past two months have seen the marital knot tighten about three more of our long-courting classmates. To date they vigorously uphold the plunge as a noteworthy improvement on the interne's life, and the protesting voices of the nays are becoming feebler and less confident.

G. W. Manning was married to Miss Marie Collins at a quiet wedding on December 14. We would be divulging military secrets if we mentioned that the event took place rather suddenly and at an earlier date than had previously been planned. George is now engaged in electrocardiography for the Air Force at the Eglinton Barracks.

G. O. Watts was married to Miss Marion McEwan on December 27 at Old St. Andrew's Church. To our knowledge the groom is not an ardent skier but Limberlost was the scene of a brief but happy honeymoon. George plans to spend his second postgraduate year in an Ontario mental hospital.

R. C. Smith took his nuptial vows with Miss Dorothy Henderson on January 24 at Metropolitan Church. Ray commenced the interne year at St. Joseph's Hospital, Hamilton, but recently has been acting as assistant to a busy Hamilton physician.

* * *

The marriages of J. V. McCutcheon and R. P. McCaffery have recently been announced. Bob left the Vancouver General to join the Air Force soon after the commencement of his interneship and Jerry at present is with the Navy in Halifax, but expects to be sailing the high seas very soon.

R. C. Ross is now with the Royal Canadian Navy aboard one of the acquired American destroyers, and N. A. Terwillegar is in Sydney, Nova Scotia, expecting to taste the salty spray in the near future.

* * *

It is interesting to note the senior appointments which have been made to date, and to obtain a glimpse of the devious paths along which we will travel.

At the Toronto General, G. E. Woodhouse, F. W. B. Hurlburt, H. B. Wallis and J. A. Traynor have received appointments in medicine. G. A. Lane and K. C. Greer have been appointed to the three-year surgical course. R. I. Probert, complete with batman to rouse him from the effects of vapours encountered, will be resident anaethestist. J. A. Weddell will be senior on Pavilion, and L. M. Hampson, after completing a year as resident aneasthetist at the Children's, will take a junior rotationship at the General.

At the Sick Children's Hospital, Taylor Statten has received the senior appointment in paediatrics, and as juniors will be G. M. Downing, Tom McConnachie, D. M. H. Prowse and R. F. Boettger. The surgical appointments have been made to W. L. C. McGill, C. H. Rorabeck, and G. C. Gray. Helen Holden will be interne on pathology.

At St. Michael's, W. H. Caven and P. F. McGoey have received surgical appointments with J. C. Emmett and J. R. Epping in medicine. E. S. McDonald will be senior in obstetrics and gynecology.

At the Western Hospital, A. L. Purdon has received a senior appointment in medicine and Bill Butt will be resident anaesthetist. G. E. P. Wilson and G. E. Cooper have four-year surgical appointments and will spend the first as demonstrators in anatomy and physiology. In pathology at the Banting Institute will be H. A. Sims, R. L. Stirrett and M. E. Hall.

J. T. R. Stewart is journeying to London where he has a surgical appointment in the Victoria Hospital.

Alf. Crossland will tell us nothing of his future plans. But more of that later,—we have already said too much.

Abstracts

R. L. MacMILLAN, Editor

Contributors D. J. Breithaupt W. E. Armour
J. G. Watt D. P. Bryce

Laboratory Procedures in Staphylococcal Food Poisoning

R. J. WILSON

Dept. of Hygiene and Preventive Medicine, Univ. of Toronto. Canadian Public Health Journal 31: 607 Dec. 1940

It is pointed out that carbohydrates utilization, chromogenicity and agglutinative characteristics are not sufficient to separate the various strains of staphylococci, which strains, being identical in the above characteristics, may vary greatly in their ability to cause food poisoning. In the investigation of an epidemic, it must be possible to differentiate strains similar to the one found in the incriminated food, and from many other sources, —throats and superficial lesions of bakery employees, and food products and baking utensils.

Four tests used for the purpose of comparing strains of staphylococci are mentioned, they are, the measurement of alpha or rabbit erythrocyte hemolysin, of beta or sheep erythrocyte hemolysin, the kitten test for enterotoxin, or food poisoning substances, and the quantitative neutralization of staphylococcus antitoxin. These are carried out on staphylococcal filtrates.

The rapid onset of projectile vomiting is the characteristic symptom in the human. The transient nature of this type of poisoning as well as the lack of food samples, case histories, and nose and throat cultures is given as the cause of inability to investigate many of the outbreaks.

D. J. B.

Thyrotoxicosis

W. A. REILLY

Amer. J. Dis. Child. 60: 79 July 1940

This is a summary of 62 children who had thyrotoxicosis, and were observed over a period of 10 years, in the last 25 years. The maximum onset was at 11-15 years of age, which was followed by 3-4 cycles of exacerbation-remission which became less and less severe and finally were fully arrested. However, it is said that a child never has a total remission, and reactivation and toxicity may recur years later in adult life.

The Basal Metabolic Rate was used, but it neither ruled the clinical impression in or out. Iodine did not seem to have much effect on the natural course of the disease. In the 62 children there were 12 definite recurrences.

33 cases were operated upon. Of these, 2 had definite recurrence of thyrotoxicosis, 2 had permanent hypothyroidism and 4 had temporary hypothyroidism. Two also developed mild permanent hypoparathyroidism and two had mild temporary hypoparathyroidism.

D. J. B.

The Pathogenesis of Paroxysmal Pulmonary Oedema

ALDO LUISADA

Formerly Professor of Medicine, University of Ferrara.

Medicine, 19: 475-497 Dec. 1940

In this scholarly article Dr. Luisada reviews the clinical and experimental conditions in which acute pulmonary oedema is seen, in order to show the inadequacy of the generally accepted increased back pressure explanation. Although in adults, most cases of acute pulmonary oedema are associated with disease of the left ventricle and/or hypertension, numerous instances exist where left ventricular failure cannot be used to explain the pulmonary oedema, e.g. trauma to the skull, operative interference with the stellate ganglia, experimental injection of silver

nitrate into the *right* ventricle, etc. It is pointed out that early in acute pulmonary oedema the blood pressure and pulse usually *rise* and that mitral stenosis, which causes a marked prolonged increase in capillary pressure in the lung, is *not* commonly associated with acute pulmonary oedema. Also many cases of coronary thrombosis die in acute failure *without* developing acute pulmonary oedema.

The experimental methods of producing acute pulmonary oedema are critically reviewed—with much of the work he was personally acquainted—and he concluded that wherever the tendency towards the development of acute pulmonary oedema was present, its development was favoured by anything causing stasis or increase in volume of the blood in the lungs. Thus the remedial value of venesection is seen. He also states that mechanical factors alone cannot cause pulmonary oedema, unless neurogenic factors are also operating.

If this neurogenic theory is accepted, acute pulmonary oedema may be grouped according to cause as follows:

A. Direct stimulation of the central nervous system as in trauma, haemorrhage, encephalitis, etc.
B. Reflex stimulation of the central nervous system:
   1. Via the heart and large arteries, as in acute distension, coronary thrombosis, etc.
   2. Via the hollow viscera—stomach and oesophagus, or irritation of the lungs or respiratory tract.

The development and course of pulmonary oedema can be influenced by mechanical procedures, and also drugs lowering the excitability of the central nervous system, respiratory system, or vegetative nerves tend to relieve acute oedema of the lungs.

D. J. B.

Primary Appendiceal Abscesses

By HICKEN AND CARLQUIST
Arch. of Surg. 42: 156 Jan. 1941

This is an excellent article for those interested in appendiceal surgery. It forms a very good review of intra-abdominal abscesses and should be of great interest to the undergraduate.

In 20% of cases of acute appendicitis there are abscesses prior to operation. The importance of draining at the right time is emphasized as is the tendency of these abscesses to be multiple with connecting sinuses. In 1 to 3% of all acute appendices pylephlebitis occurs. In such cases the mortality is about 85% and the only hope lies in early exploration with drainage of regional abscesses and ligation of the involved venous radicles and in chemotheraphy.

In no case is there sufficient urgency to prohibit proper preparation of the patient with restoration of his fluids and decompression of the gut. Finally the value of post-operative care is stressed with the use of chemotheraphy to prevent complications.

D. P. B.

The Diagnosis and Treatment of Hyperthyroidism

J. H. MEANS, M.D.
Boston, Mass., U.S.A.
C.M.A.J., Dec. 1940

The term hyperthyroidism implies that too much thyroid hormone is being projected into the bodily economy. This may occur when thyroid hormone is given therapeutically, when the thyrotropic hormone of the pituitary is being administered or when toxic goitre occurs spontaneously. The last of the three is considered in this article.

A history of trembling, weakness, palpitation of the heart, increased appetite, loss of weight, and sensitivity to cold makes one suspect the disease. The combination of increased appetite and progressive weight loss is almost pathognomonic. If one also finds enlargement of the thyroid or any of the eye signs characteristic of Grave's disease, the probability of toxic goitre in the patient becomes great. Finally, if there is a persistently elevated basal metabolic rate, which falls when iodine is given and rises again when iodine is withheld, the diagnosis is established.

The differential diagnosis is from simple goitre with nervousness of neurotic origin, rarely from leukemia in which the metabolic rate is raised, and more rarely from tuberculosis. It may be difficult to separate heart disease of some other origin from toxic goitre with marked cardiac symptoms.

There are three important methods of treatment, all aimed at reducing the activity of the thyroid gland. They are: operative removal of a portion of the gland, irradiation of the gland, and administration of iodine. Means believes that the treatment of the future will be by hormones.

Iodine—The thyroid gland has a great affinity for iodine, and this affinity is greater in toxic goitre. Iodine is used therapeutically in three ways; first, preparation for operation; second, for thyrotoxicosis persistant after operation or recurrent thyrotoxicosis; third, as a definite form of treatment by itself. As a preparation for operation iodine is obligatory. If instead of abolishing thyrotoxicosis, operation merely reduces it, then the continued administration of iodine may further reduce it and hold the symptoms in complete or nearly complete abeyance, until the time when the disease reaches a natural cessation. Iodine alone is usually only a palliative measure, but it may be curative in certain selected mild cases, particularly in women who develop the disease at the time of the menopause. In some of these cases it may be possible to omit the iodine after six months of continuous treatment without having a return of symptoms.

Surgical Procedures—Subtotal thyroidectomy following the preoperative use of iodine is still the best method of treatment.

Irradiation—Means uses X-ray treatment for certain uncomplicated cases of Grave's disease, and for recurrent toxic goitre. Instead of resorting to a second operation if the symptoms return following subtotal thyroidectomy, he uses X-ray and finds that further surgery is unnecessary.

A disturbance that produces a marked acceleration of basal metabolic rate may be expected to often cause grave nutritional results, and there is good reason to believe that these patients suffer from vitamin B deficiency, other vitamin deficiencies and some degree of hypochromic anemia. For these patients iron in full doses and yeast may prove beneficial.

Hyperthyroidism may aggravate preexisting conditions such as diabetes mellitus, Addison's disease, or the cardiovascular apparatus, the liver or the psyche may break down under the added strain. Getting rid of the thyrotoxicosis is of great benefit to these cases.

There is a small group of cases in which the main problem is ophthalmological. The eye signs may progress despite the fact that the thyrotoxicosis is entirely relieved. The pituitary plays some role here. In the more severe cases called malignant exophthalmos there is marked swelling of the orbital muscles and intraorbital tissues. Oedema is the main feature, even more than protrusion of the eyeballs. Evidence is accumulating that treatment by thyroid extract is beneficial and thyroidectomy is injurious to these cases. The thyroid may act by inhibiting the anterior pituitary or it may actually cause the fluid contents of the orbit to decrease. If there is progression despite thyroid extract then intracranial decompression of the orbit is required to save the vision.

J. G. W.

"The Blood Sedimentation Test as a Routine Diagnostic Procedure"

E. B. AGNOR, M.D.

Annals of Internal Medicine, Nov. 1940

The sedimentation test was originally a test for pregnancy; it was dropped, and later revived. This article is the report of a series of 2,063 cases; the original technique of Westergren was used, with citrated blood.

There were four classes of reaction:

I. Normal, 0-15 mm. fall in one hour. II. Borderline, 16-20 mm. III. Slightly elevated, 21-30 mm. IV. Definitely elevated, 31 or more mm. in one hour.

It was found that, of all cases with

definite organic disease, 43.9% had elevated rates; of those without, 4% showed an increased rate. Of the former, in those suffering from a disease usually accompanied by an increased rate, the rate was elevated in 73.5%; there were proportionately more males than females in this group. There were 11.6% with increased rates in the group suffering from diseases not usually characterized by such an increase.

It was also found that it was unnecessary to correct for anaemia, and that the white count and the sedimentation rate did not parallel one another to any significant degree.

W. E. A.

"Mucous Colitis: a Delineation of the Syndrome with Certain Observations on its Mechanism and on the Role of Emotional Tension as a Precipitating Factor"

B. V. WHITE, M.D., AND
C. M. JONES, M.D., F.A.C.P.
Annals of Internal Medicine, Nov. 1940

Conclusions are drawn from a series of 60 patients, of whom 57 were studied psychologically, with regard to the emotional factor concerned and their personality type. The subject is discussed under the headings of Clinical Syndrome, Mechanism, and Role of Emotional Tension.

The diagnosis was confused with sixteen other conditions, of which five were gastrointestinal conditions. The most usual cases had, as outstanding symptoms, constipation, occasionally diarrhoea, and some form of abdominal pain. The stools were mushy, or took the form of hard pellets. There was usually associated autonomic instability, evidenced by such signs as dermatographia. The changes in the rectosigmoidal mucosa were explained by activity of the autonomic nervous system, by drugs taken for relief, or both. There was commonly nervous tension most often caused by anxiety, resentment, or guilt. 50% of the cases were the rigid type of thinker, such as are found in the obsessive-compulsive states. The condition is a definite somatic response, not psychologically determined, and not gainful like hysterical conditions.

W. E. A.

Medical Management of Fractures

By NELSON AND NELSON
J.A.M.A. Jan. 18, 1941

What are the factors concerned with the metabolism of bone and how may they be combined to form a basis for the medical treatment of fractures of all kinds and especially those cases of malunion? This problem is discussed on the basis of over one thousand cases of fractures including cases of non-union, delayed union, those complicated by osteoporosis and diseases such as Paget's, fragilitas ossium and others. Animal experimentation is also carried out.

The authors have included in their paper the discussion of serum calcium and phosphorous and the effects of parathormone and vitamin D on bone metabolism. They have combined these factors in a system of medical treatment which has not resulted in failure of primary union in a fresh fracture since its inception. Also they have been able to regenerate delayed union in many cases referred to them.

The basis of their treatment lies in blood chemistry examinations of all fracture cases with an object to maintain a serum calcium level of from 10.5 to 12 mgm. % and a serum phosphorous of from 3.5 to 4 mgm. %. Medical treatment for a low serum calcium is recommended as this condition exists in 85% of all fracture cases.

D. P. B.

Calcium in the Coagulation of Blood

By A. J. QUICK
Amer. Journ. of Physiol. Dec. 1940

The author describes a series of experiments concerning the effect of calcium on clotting. He uses a system containing a fixed level of prothrombin, an excess of thromboplastin and added calcium as the

variable. It was shown that active prothrombin contains calcium and may be represented by the formula prothrombin-calcium. The oxalating of this organic-aly combined calcium, to render the prothrombin inactive and so prevent clotting, was shown to be slow unless a large excess of oxalate was used. This reaction was shown to obey the mass law. Of practical significance is the fact that if a large excess of oxalate were not added to freshly drawn blood some thrombin would be formed producing fibrin threads which would make the blood unfit for transfusion. Another important observation was made. The clotting time of normal plasma, to which thromboplastin is added, is the same as that of oxalated plasma to which thromboplastin and a fixed quantity of calcium chloride is added. The latter procedure will be recognized as the laboratory estimation of the serum prothrombin level. Thus the addition of oxalate and then calcium chloride does not introduce variables effecting the clotting time and so the prothrombin level estimated in this manner may be taken as valid. R. L. M.

Volkmann's Ischaemic Contracture

By D. GRIFFITHS

Brit. Journ. of Surg. 28: 239 Oct. 1940

The three theories of the pathogenesis of Volkmann's ischaemic contracture are discussed and the author presents convincing evidence that the primary cause is arterial injury. The ischemia is caused by a spasm of the collateral circulation. The pathological changes observed in the damaged arteries and infarcted muscles are described. The most important sign of onset of the contracture is the absence of radial pulse. Pain and numbness of muscles of forearm usually indicate irreversible damage. The treatment of this condition is discussed. Prevention is stressed. Wherever there is a weak or absent radial pulse, following a fracture, steps should be taken to improve the pulse. Gentle manipulation, surgery and anti-spasmodic measures all have their place as is indicated in this very interesting article. R. L. M.

End Results of the Injection Treatment of Hydrocoele

By EWELL, MARQUARDT, SARGENT

Journ. of Urology, 44: 741 Dec. 1940

After the aspiration of all possible fluid from the hydrocoele quinine urethane solution was injected. This was repeated several times if necessary. The solution is not strongly irritating and no untoward results were produced. Its use is contra-indicated where the hydrocoele is congenital or infected. Of 165 cases treated in this manner there were only 4.3% failures and recurrences. This figure compares favourably with results obtained with surgery. A striking advantage of this method is the fact that the patients are ambulatory.

R. L. M.

Experimental Coronary Sinus Obstruction

By H. F. ROBERTSON

Surgery 9: 1 Jan 1941

Vieussens and Thebesius have demonstrated small channels that join the cavities of the heart to the coronary arteries and veins. The author investigated the possibility that in coronary artery occlusion, blood might pass from the cavities along these channels to nourish the heart muscle. Ligation of the coronary sinus causes these channels to become dilated. If following this the coronary arteries were occluded, it was hoped that these channels would function to good advantage and sustain the heart. However, this was not substantiated by the experiments described in the article. No blood flow, sufficient to nourish the heart could be demonstrated passing back along the Thebesian veins from the ventricles. In fact ligaturing the coronary sinus predisposed to a wide necrosis of heart muscle in a number of cases. These results might have been anticipated from a consideration of intra-ventricular pressures. Only for a very short time in the cardiac cycle are pressures suitable for blood to pass from the heart cavity to the muscle by the veins of Thebesisus.

R. L. M.